The Heart of Mitral Stenosis

Drawing by E E Hoff

HENRY FORD HOSPITAL

International Symposium on

Cardiovascular Surgery

HENRY FORD HOSPITAL

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PREFACE

During the summer of 1954, the staff of the Henry Ford Hospital, with the encouragement of the executive director, Dr Robin C. Buerki, decided to sponsor an international symposium on the subject of surgery of the heart and great vessels With realization of the importance of applied physiology and diagnostic methods, the following title was chosen for a symposium to be held on March 17, 18 and 19, 1955 Cardiovascular Surgery: Recent Studies in Physiology, Diagnosis and Techniques The local program committee sought and obtained the assistance of four eminent authorities from outside the staff of the hospital. Grateful acknowledgment is made for the valuable advice of Dr Richard J Bing of Birmingham, Alabama, representing the field of applied cardiac physiology, Dr Stanley Gibson of Chicago, pediatric cardiologist, Dr Emile Holman of San Francisco, and Dr Michael DeBakey of Houston, Texas, surgeons who have had particular interest in cardiovascular disease.

The program committee met in Detroit and carefully considered the material which appeared to be essential for discussion It was thoroughly understood that the symposium was not to be a postgraduate course in which subject matter well known to certain specialists would be reviewed, but rather it would be an opportunity for the presentation of really new and perhaps controversial work. It was decided to spend one day on the problems of congenital heart disease, another on acquired heart disease, and a half day on surgery of the aorta and other arteries

Invitations to participate in the symposium were sent to sixty workers in the field, many of whom resided in countries other than the United States The response on the part of the invitees was nearly 100 per cent in the affirmative. The tentative program was changed from time to time to incorporate certain late suggestions from the participants and others Two months before the meeting, the final program was sent to a selected list of teachers and workers to whom the symposium material would presumably have special interest and value Several weeks before the date of the symposium, there were more applications than there were seats in the Clinic Building Auditorium. Thereafter, the local committee was obliged to send letters of regret. The final registration list contained the names of 478 doctors, from 35 states and the following other countries Argentina, Australia, Brazil, Canada, Chile, Colombia, Costa Rica, Cuba, Denmark, Great Britain, France, Holland, India, Italy, Jordan, Mexico, New Zealand, Norway, Puerto Rico, Sweden, Switzerland and Venezuela

This volume contains the material which was presented during the two and one-half days of the symposium. The participants responsible for the prin-

cipal papers have cooperated by furnishing manuscripts which are amply illustrated. The editor acknowledges the excellent work of Mrs. Charlotte Emmons of the Master Reporting Company who accurately stenotyped the discussions, many of which were given in the delightful accents of the foreign participants

The editor is grateful for the cooperation and assistance so freely provided by the staff of the W. B. Saunders Company.

A final word of appreciation on behalf of the program committee should be given to those whose work is reported in this volume—the participants, who left their important teaching work and busy practices to form this symposium.

CONRAD R. LAM

Detroit, Michigan

FOREWORD*

By Sir Russell Brock

I must first thank you for the great honor you have done me in asking me to be your guest speaker at the dinner tonight marking the occasion of this great international symposium. I little knew what I was letting myself in for when Conrad Lam spoke to me about this meeting in September last at the International Cardiac Congress in Washington. He told me that it would be "just a small friendly crowd of people interested in cardiovascular surgery chatting together round a table." Now I find myself addressing a crowd of between 400 and 500 of you, and as I survey this large throng my pride in being asked to speak to you is modified by the difficulty of the occasion. While I stand before you on this lonely pinnacle I am reminded of the story of Sir Edmund Hillary's reply when he was asked his emotions as he stood on the top of Everest. Hillary said he experienced three emotions the first was relief at having reached the summit, the second was pleasure at having achieved the goal so long sought, and the third and most powerful emotion was an intense desire to get down again as quickly as possible.

I am especially grateful to you for asking me to address you as I realize that, being a foreigner, the honor is so much the greater At the same time I am very happy to be able to speak to you as this is far from being my first visit to the United States, not only have I many, many friends here, but I have looked upon it as my second surgical home ever since I was privileged to work with Professor Evarts Graham in St. Louis nearly twenty-five years ago The impressions and inspiration I gained then as a young surgeon visiting Barnes Hospital and numerous other great surgical centers in your country were such a powerful influence that they had a permanent effect on my surgical thoughts and career I am particularly proud of the certificate I hold that proclaims I am a visiting surgeon to Barnes Hospital. I am equally proud of a similar certificate giving to me the rank of honorary surgical consultant to the Johns Hopkins Hospital in 1949 after I had spent a wonderful and happy month there as visiting professor

If I had not known beforehand how important and successful this symposium was to be I should have begun to guess as soon as I got on the 'plane to cross the Atlantic. As fellow passengers I found several friends and surgical acquaintances from Great Britain and from other European countries I think one of the most significant testimonies to the success of the symposium is exemplified in the way so many men have come such long distances from

* Text of an address delivered at the dinner meeting of the Henry Ford Hospital International Symposium on Cardiovascular Surgery, March 18, 1955

far off countries, and at great personal expense, to attend our deliberations. I think they have been well rewarded for their trouble by the excellence of the symposium and I am sure I shall be speaking for all of you when I accord not only our sincere congratulations but also our best thanks to the organizers for the truly magnificent job they have done Chief among these must be our friend Conrad Lam, who has obviously been the inspiring genius in the matter.

In addition to the many who have come simply to listen to the presentations and discussions, and these include many names famous in thoracic medicine and surgery, there is a formidable list of participants invited to present papers or to take part in the panel discussions It is impossible to mention all the distinguished names here today, but as my eye traveled down the list of participants I felt I must say a few words about some of them I mention them in simple alphabetical order and I first see Dr. E C. Andrus, recently President of the American Heart Association, whom I last met while he was shaking hands in the long receiving line at the Washington Congress, he was a friend I first met earlier at Baltimore in 1949. Henry Bahnson, another friend from Baltimore, came to Guy's in 1947 as first assistant to his distinguished chief, Alfred Blalock, who honored us by spending a month there as exchange professor and by demonstrating to large crowds of eager visitors the technique of the new Blalock-Taussig operation We all felt sure that Henry would go far and this feeling has been justified, as we all know when we hear him speak of the magnificent work he is doing in this difficult task of resection of aneurysms of the aorta.

There are several immortals in the field of cardiac surgery here tonight, men who have made great fundamental contributions, always prominent amongst these pioneers will be Charles Bailey In addition to his intrinsic contributions in cardiac surgery I feel that by his very enthusiasm and continued effort he has perhaps done more than anyone else in this country to press forward the acceptance and development of cardiac surgery

Claude Beck, the veteran of cardiac surgery, a man who has devoted a lifetime to the study of the surgical relief of cardiac ischemia, delighted us all by his brilliant speech today on the treatment of cardiac arrest, another subject to which he has made great contributions and with which his name will be forever associated

Dr Bigelow, from Toronto, who has pioneered the fundamental work in hypothermic states

Richard Bing who gave us the excellent opening paper yesterday on cardiac catheterization and who, in addition to being such an able investigator, is such a delightful companion and humorist.

Gilbert Blount—there is a fighting physician for you; right up in the front line of the operating room where many more cardiologists could be with benefit.

Denton Cooley who with his senior, Michael DeBakey, presented his magnificent work in the resection of aortic aneurysms, and especially a phenomenally large number of abdominal ones, was another friend I made at Baltimore We were then very pleased to have him for six months in London where he worked as first assistant on my unit at the Brompton Hospital

In Clarence Crafoord we see our true international surgeon, always to be met at meetings and symposia all over the world. We remember his earlier success in pulmonary embolectomy—work never equaled—and also his later association with the introduction of the radical treatment of aortic coarctation

Charles Dubost represents our Paris friends, there we have an able, clever, courageous and courteous surgeon.

Jesse Edwards, whose observations on cardiovascular pathology have been so helpful.

Frank Gerbode, a clear minded, progressive surgeon who, as well as doing excellent practical cardiovascular surgery, is leading a fine team of young men in surgical laboratory investigations

John Gibbon who has devoted so many years to the development of the artificial heart-lung and has been responsible for one of the earliest successful cases treated by this method

Dwight Harken is another Bromptonian, he spent a whole year with us at the Brompton before the war His great contributions to cardiac surgery we all know, but I am happiest of all in remembering his work at the U S Army Hospital near Cirencester during the war I had the privilege of visiting him there and of seeing some of that remarkable series of 11 cases of open cardiotomy for removal of an intracardiac foreign body with 11 successes A brilliant performance which presaged his present success

Dr Emile Holman, that doyen of cardiac surgeons, remains as active and vigorous as ever I especially enjoyed his recent presentation of the problem of post-stenotic dilatation of vessels as exemplified by pulmonary valve stenosis

Charles Hufnagel earns admiration from us all in his brilliant work on the difficult problem of aortic regurgitation. Even if his plastic valve is later superseded it must mark an historic stage in the evolution of valve surgery

I have already mentioned the debt we owe to Conrad Lam for his masterly organization of this symposium.

To Walton Lillehei we accord the high spot of this meeting; nothing could be more stirring, exciting and significant than his presentation of the work of the Minneapolis team on cross circulation. Here he has truly shown us that open, definitive intracardiac surgery is here, now, today I for one have a special interest in his work as he has demonstrated so beautifully and convincingly what I have maintained for some time: that direct relief of pulmonary stenosis alone can lead us to the complete cure of Fallot's tetralogy

Dr Lind has delighted and impressed us with the magnificent radiologic technique we have learned to expect from Stockholm.

Willis Potts, in addition to his services as toastmaster this evening, deserves the thanks of many of us for the beautiful instruments he has designed and provided for our greater comfort and efficiency in these difficult cardiovascular operations, we thank him sincerely and record our admiration of his pioneer surgical work.

The two Robbs, one with two b's and one with one, are both from outside this country Rob, we hear tomorrow when he will tell us of his pioneer work in London on vessel grafting He organized the earliest blood vessel bank in England. As Professor of Surgery at St. Mary's Hospital, he is asso-

ciated with a hospital whose Rugby football team has been successful in defeating my own hospital (Guy's) for many years until this year; only last week in fact, after a replay owing to a drawn game in the first encounter, Guy's succeeded in winning by scoring a touchdown in the last minutes of extra time!

Douglas Robb is the leading thoracic surgeon in New Zealand, as an old friend I have been glad to see much of him during these last few weeks

Henry Swan has delighted many of us who have visited his clinic at Denver and seen and admired his splendid work on hypothermia. We have even envied his superb long series of cases successfully treated with this technique

I said I was taking these names in alphabetical order, it enables me to end with Dr Helen Taussig whose inspiration and carefully ordered conclusions on the basic factors in cyanotic heart disease enabled her to inspire and guide the happily convenient technique of Alfred Blalock

Although my lot seems permanently cast as an antagonist of her work, I hope and indeed know that she and Alfred really know how much I admire their work, which was perhaps the greatest of all the stimuli that started off the chain reaction that has progressed to what we have heard these last two days Dr. Taussig must indeed be proud when she reflects on this and we rejoice with her.

SURGICAL FRONTIERS

And what remarkable things we have listened to in the last two days, what astonishing technical development in this new field of surgery. It seems certain that we have now reached the last anatomic development of surgery, prophecies can be very dangerous but it is difficult to visualize any other anatomic field that remains now that the interior of the heart has been successfully reached This is not to suggest that we have come to the end of surgical advance, future developments, however, will have to be along pathophysiologic pathways For instance tomorrow we discuss the surgery of arterial diseases to remind us of the pioneer work of Carrell in vessel grafting, more lately revived and re-introduced by that great surgeon Robert Gross, perhaps we shall see surgery extended further by the grafting of other tissues and even of organs, although it would seem that this will not be biologically possible. The delay or relief of the degenerative diseases also offers great prizes to the surgeon, and of course there always remains one of the great natural problems of surgery—the influence and control of the processes of wound healing.

CARDIAC SURGERY—A NEW SURGICAL INDUSTRY

Although in this gathering I speak to the converted, I must mention a common misconception that cardiovascular surgery is something new and glamorous It is true that it is relatively new and that it may be glamorous, certainly it may be exciting and is often of high news value, but because its development has been rapid it does not mean its foundations are shallow or ephemeral In their early days radio or television could have been said to be new, exciting and sensational, but their rapid success was certain because they provided something that was lacking Cardiac surgery has achieved rapid success for the same reason, it has successfully supplied something that was

not available before—relief to a large group of otherwise hopeless and incurable invalids Here is no flash-in-the-pan but a strong new surgical industry This success and stability is emphasized not only by the many who are attending this symposium but also by the many who tried but could not find a vacancy

THE CHANGING PROBLEM

This large gathering of so many interested in thoracic surgery from the United States and from abroad also shows the change in the pattern of thoracic surgery that it has been possible to observe during recent years I so well remember when I visited America in 1949, a few thoracic surgeons were keenly interested in cardiac surgery, but most had only that interest that any intelligent man has in the developments in his own speciality or profession, most did not foresee that they would personally become involved.

Now all is different, almost all thoracic surgeons are keenly interested in taking part in the new field of work. The whole pattern of thoracic surgery is changing; bronchiectasis is a lessening disease, lung abscess is rarely a surgical condition, tuberculosis is fast dying out and in any case is profoundly altered in its relation to surgery by the new antibiotics and chemotherapeutic substances, bronchial carcinoma alone increases in incidence and importance.

THE PLACE OF THE SURGEON

This brings me to another feature of our professional life that has always given me much thought, namely the relation of the surgeon to the physician.

Here again I speak to the converted because everyone here recognizes and indeed has shown in this symposium, the proper cooperation that must exist between physician and surgeon, that only by close team-work can efficient work be done.

However, this attitude is not always seen, or not its full happiness everywhere, perhaps this absence is less in the United States than in England, but it doubtless occurs even here. In any case this is meant to be a dominantly *surgical* symposium and the mnay surgeons here will doubtless bear with me and the physicians will not be offended by my observations The surgeon receives so many wisecracks directed at him that there is no harm in his saying a few words in his own defense.

I think the tradition of the surgeon being a somewhat lower form of life than the physician is rather stronger in England, whenever I think of this I am reminded of a scene that you may see any day in London. If, sometime in the early evening, you walk from the Bank of England towards Westminster and Buckingham Palace you will be certain to meet a detachment of Her Majesty's Foot Guards in full uniform with fixed bayonets, marching with an officer at their head with a drawn sword. They make a magnificent spectacle and they march along at a smart pace ignoring traffic lights and everything. If you inquire you may be told by the informed that they are the 'Bank Guard' , they are on their way to guard the Bank of England. Now, in 1780, very soon after George III had ceased to be the last King of America,

there was a civil disturbance in London led by Lord Gordon and called the Gordon Riots. As the mob threatened the Bank of England protection was sought and orders were issued that a detachment of Guards should be sent to watch over the Bank Well, the Gordon Riots are long past but the guard still marches to its duties at the Bank because unfortunately no one remembered to cancel the order that they should do this every night. Of course if anyone tried to stop it now it would probably lead to another riot; apart from the fact that the guard itself has a vested interest in that the men get free beer and their officers free wine, it is now part of the tradition of England.

At the same time as the Gordon Riots, that is in the latter part of the eighteenth century, another English tradition was strong and has persisted nearly as strong to this day. That is the peculiar relationship between physician and surgeon. Then there was reason for it, the physician was alone likely to be the trained scientist with a university degree and the ability to think for himself. The surgeon was, in general, an ignorant fellow, a pure follower of a craft and with little or no power of original thought. In London on the one hand was the ancient Royal College of Physicians, on the other was the Barber-Surgeons Company, originally founded by Henry VIII and continuing in increasing disharmony until the final separation of the two groups in 1745. This separation arose from the intolerable association with the unskilled barbers forced upon those surgeons who were beginning to realize that their craft must become a science and must follow the laws and rules of other learned callings. Cheselden, for instance, was one of these earlier scientific surgeons and was largely instrumental in securing this separation so essential to the proper development of surgery. At about the time of the Gordon Riots John Hunter, the first of the really great scientific surgeons, was in his prime It has been claimed that to him belongs the greatest credit for putting surgery on the right scientific road The proper organization of surgery and the education of surgeons on a scientific basis certainly date from this time. After an uneasy period the Surgeons Company was replaced in 1800 by the Royal College of Surgeons, comparable with the Royal College of Physicians, and we then began to see the surgical giants of the nineteenth century, culminating in the immortal Joseph Lister and the complete transition to the scene of modern surgery.

However, by tradition in the eighteenth century the surgeon was a lower form of life than the physician, and by tradition he tends to remain so in the minds of some It is true that in England we pay lip service to this tradition, thus we still call our surgeons Mr instead of Dr. as the earlier surgeon was rarely or never an M D, but we reject it utterly when it affects our work, and this is the real reason for my discussing such a touchy subject. In cardiology there can be no doubt that this tradition of the inferiority of the surgeon has affected the development of cardiac surgery and, although not to such an extent as formerly, is still affecting it I know that this does not apply to anyone here, present company is always excepted, but it certainly continues to do so elsewhere I know that in the past the development of cardiac surgery was delayed and hindered by this tradition because I encountered it so much in

my early efforts to develop the surgery of mitral stenosis, I am sure many of my surgical confreres had similar experiences That phase is now largely a thing of the past in mitral valve surgery, but even today it is illuminating to hear at times from patients of their adventures in trying to reach a surgeon to obtain the relief that surgery alone can offer them. I must say that the great pressure to aid the development of mitral valve surgery came not from the doctors but from the patients who, in their frustrated desires to obtain help in their disability, brooked no interference. I fully realize, of course, that the natural conservatism and caution of the physician still serves a useful and at times even an essential purpose

We recognize the great debt we owe to our physician colleagues for their great contributions now and in the past, and for the great help we daily receive from them, but all must recognize that the physician and the surgeon now stand on an equal footing The traditions of the eighteenth century are ghosts of the past.

I have never been able to understand why the surgeon should continue to be thought inferior We are conceived in the same way, born in the same way, eat the same food, drink the same drink, go to the same schools and colleges, attend the same medical schools and the same clinical instruction and graduate in the same way Whence and where derives this subtle difference between the two? I can only conclude it is something in the very nature or atmosphere of the Royal College of Physicians, perhaps a cloud of some special canonizing dust falls upon its habitués.

SURGERY AND CARDIOLOGY

All of us here recognize, and indeed it is imperative to recognize, that surgery is now and forever a part of cardiology It has, in fact, caused a true renaissance in cardiology which recently had seemed to be progressing slowly But surgery not only gives an equal contribution to cardiology but it can also lead and direct, it can and does form an integral part of modern cardiology which certainly extends right into the operating room where the heart is exposed, examined and assessed in a way hitherto impossible, quite apart from the therapy that can be carried out.

There is another tradition in England exemplified by the story of King Canute. You will remember that this great Danish monarch reigned in England in the eleventh century and his power was so great that his courtiers told him that even the waves and the sea would obey his commands King Canute ordered his throne to be carried to the edge of the incoming tide and demonstrated by scientific experiment that his commands had no effect on the waves, that their onward passage was inexorable. There are still a lot of people who imagine that they can hold back the irresistible forward march of cardiac surgery by negativism. It is no more possible to do this than to hold back the tide and the waves of the sea.

In addition to actual therapy what can surgery contribute to cardiology, or rather what has it contributed? First is a revision of the standards of diagnosis Although we owe much to the diagnostic ability of many cardiologists

and to the great and basic contributions by them, one cannot but be impressed with the low standard of diagnosis one often meets; especially is this so in congenital heart cases. In the past errors in diagnosis were less important, except in so far as a mistaken diagnosis of severe heart disease where none existed might ruin a healthy patient's life The interpretation of physical signs, especially cardiac bruits, was often in the nature of a pleasant parlor game The physician taught that certain noises meant certain diseases, the student heard and recounted his information at the examination, was duly approved and went back to pass on the same teaching. No harm was done and very often no good

Now, however, it is different When the question of an operation arises diagnosis must be exact, and until we achieve a high standard of diagnosis no high standard of surgery is possible. Cardiac surgery today insists on very high diagnostic standards. It recognizes also that even when every care has been used a full and complete diagnosis is sometimes impossible until the heart is exposed at operation In other words cardiology today extends right into the operating room. Surgery supplies a great additional help to cardiology in its ability to allow study and observation of the living heart as opposed to the dead one. So many things are different when we study them in the live, functioning organ.

Another great help that surgery can bring to cardiology is connected with certain of the basic functions of surgery. Almost from time immemorial the surgeon has been concerned with certain simple things such as the control of hemorrhage, the mending of broken bones, the drainage of abscesses and the relief of obstruction The surgeon knows full well that an obstructed viscus or duct must be relieved and he is, moreover, fully aware of the part played by muscle power in the physiology and pathology of obstruction. He knows, for instance, that the surgeon's duty is to relieve an obstruction however unfavorable the problem or circumstances may appear to be, unless the patient is moribund This principle applied to difficult cardiac problems will often simplify and clarify many situations that otherwise seem hopeless The cardiologists, more so than any other medical specialists, have in the past been very little exposed to the influence of the surgeon The surgeon, by his special training, can contribute much of great value, not alone in details but also in principles.

THE FUTURE

I have dealt with the past and the present—what of the future? We need a steady supply of new ideas, the courage to apply them and tenacity of purpose in our difficulties Difficulties are in plenty all along the way, all of us know the heartache and despair that can beset us and the black moments that come from a series of failures. In these black moments it is essential for the surgeon to hold to his plans and policies and program, provided he has confidence that he is in the right. I remember at an early stage of our development of the surgery of mitral stenosis we had had four successive deaths in our women's ward Despair stalked before us and everyone's morale was low. I remember saying to my team that we could do only one of two things, give up or go on;

that it was impossible to give up as we were certainly in the right; the only thing, therefore, that we should do was to go on. We did continue and had 30 consecutive successful cases

In this need for tenacity of purpose I am reminded of Drake s prayer Although Francis Drake was English, and in fact very specially English, he has some connection with America. You may remember that on his famous voyage round the world he sailed up the west coast of America, what is today California As is often the case it was foggy and so he sailed past the Golden Gate. Perhaps if it had not been foggy and he had sailed through the Golden Gate into San Francisco Bay, who knows that he might not have changed the history of the world!

His so-called prayer was not spoken as such by him but was composed from a sentence in a letter he wrote when he lay outside Cadiz harbor before going in to set fire to and destroy the Spanish ships

"O Lord God, when thou givest to thy servants to endeavour any great matter, grant us also to know that it is not the beginning, but the continuing of the same unto the end, until it be thoroughly finished, which yieldeth the true glory"

That should be our guiding thought and it has certainly been exemplified in what we have heard in this symposium.

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CARDIAC CATHETERIZATION

R. J. BING (*Birmingham, Ala.*)

I am greatly honored to open this Symposium on Cardiovascular Surgery. Since I am not a surgeon, I derive great comfort from the subtitle of this Symposium: "Recent Studies in Physiology, Diagnosis and Techniques" This subtitle shall be my guiding light, and I shall therefore devote this paper on catheterization primarily to the advances in our understanding of the physiology of the cardiovascular system which have been made through the technique of catheterization I use the term "technique" advisedly, because, after all, the introduction of a tube through a vein and thence into the heart, or into the renal or hepatic vein, is nothing but a technique; it depends to a great extent on the individual investigator whether it remains solely a technique or becomes a tool for physiologic investigation. It is like playing the piano—there are many people who can move their fingers around the keyboard, but few who can express the spirit of music.

CONTRIBUTIONS OF CATHETERIZATION

In discussing the contribution of catheterization to our understanding of cardiovascular physiology, we must first of all mention the measurement of the cardiac output After Forssman's catheterization of his own heart, it was Klein in Prague who used catheterization first to determine the cardiac output in man by means of the Fick principle [1,2] Everyone is familiar with the impetus which Cournand and his school have given to this technique [3] Although in recent years some justifiable doubt has been cast upon the accuracy of the Fick principle, it should be said that the error is probably not as significant as was first believed by Dr Visscher and his group in Minnesota [4]

PULMONARY CIRCULATION The catheter has given us great insight into the physiologic adjustments of pulmonary circulation. One of the most important concepts that have arisen from such studies is the finding that the pulmonary artery pressure remains stable in the face of wide variations in blood flow [5] The Bellevue group found that when the blood flow through the lungs, or even one lung alone, increases up to 2½ times the range of basal flow, the blood pressure in the pulmonary artery does not increase significantly [6] Beyond this level of blood flow the pressure rise is rapid. If the capacity of the pulmonary vascular bed is reduced, the curve depicting low pressure relationship is obviously different, and smaller increases in flow are associated with greater arterial pressure rises In pulmonary disease in which the pulmonary

This work was supported by grants from the U S Public Health Service, the American Heart Association, and the Life Insurance Medical Research Fund

resistance has become fixed, a rise in pulmonary blood flow is accompanied by a disproportionately large increase in the pulmonary artery pressure. However, a warning should be inserted. It has justly been pointed out by Cournand that before interpreting a pressure change in the pulmonary circulation as related to variations in "arteriolar" resistance, one must first investigate whether the volume capacity of the pulmonary bed has remained normal. A rapid shift of blood from the left heart can greatly influence the pulmonary artery pressure without changing the pulmonary vascular resistance. This is illustrated in the tests dealing with the effect of digitalis on the cardiac output, the stroke volume, and the pulmonary artery pressure in patients with left-sided failure.[7] The small pulse pressure in the face of an increasing stroke volume presumably indicates that blood has been displaced from the lung as the performance of the left ventricle improved, as a result, the systolic ejection of the right ventricle takes place into the less filled pulmonary system

The whole question of pulmonary vascular resistance could be reexamined in man as a result of the catheterization studies The work of Hellems has made it possible to estimate the pressure in the pulmonary veins [8] Although it is at the moment recognized that the wedge pressure, or the pulmonary capillary pressure, is extremely slow in following phasic changes in the left side of the heart, it is gradually recognized that the pulmonary wedge pressure permits an estimation of pressure in the pulmonary veins and thus enables us to calculate the resistance across the pulmonary bed. Using these measurements, it was found more than eight years ago that in many types of congenital heart disease the resistance in the pulmonary vascular bed is increased.[9, 10] This is now an old story, but I remember still the excitement we experienced at finding the tremendous pulmonary hypertension in patients with Eisenmenger's disease and in some patients with large left-to-right shunts This has led to a reconsideration of the problems of the relationship between anatomic changes in the pulmonary vascular bed and alterations in blood flow These questions will be dealt with in a subsequent portion of this Symposium. Finally, catheterization has made possible an investigation of the effect of hypoxia on the pulmonary circulation. Regardless of whether hypoxia results in fluid displacement into the pulmonary circulation or in active constriction of the pulmonary vascular bed, the catheter has at least established the fact that, in man, hypoxia results in pulmonary hypertension.[11]

HEPATIC BLOOD FLOW The catheter has also made possible the estimation of hepatic blood flow in man, through catheterization of the hepatic vein and the use of the Fick principle by Bradley [12] Using this technique, Myers found that by plotting in normal individuals, in patients with cardiac failure and in individuals with severe anemia, the arteriovenous oxygen difference against the hepatic flow, an exponential curve is obtained[13], this illustrates that in these groups the splanchnic oxygen consumption remains constant, and the hepatic flow varies inversely with the arteriovenous oxygen difference In contrast, results from catheterization of the coronary sinus have indicated a very different relationship between the myocardial oxygen extraction and coronary blood flow the oxygen consumption of the heart is almost exclusively regulated by variations in coronary flow [14] Naturally, this compensatory

rise in splanchnic flow in the presence of diminished arteriovenous oxygen difference across the splanchnic bed has its limits; for example, in severe hemorrhage the oxygen consumption falls because the flow cannot keep up in compensating for the diminished splanchnic arteriovenous oxygen extraction and the splanchnic oxygen consumption declines. Exceptions to this rule have also been discovered in thyrotoxicosis, where the hepatic blood flow is not significantly altered, but where the hepatic arteriovenous oxygen difference and the splanchnic oxygen consumption are markedly increased.[13] Again, this is a different situation from the heart and the brain, when the oxygen consumption in thyrotoxicosis is within normal limits.

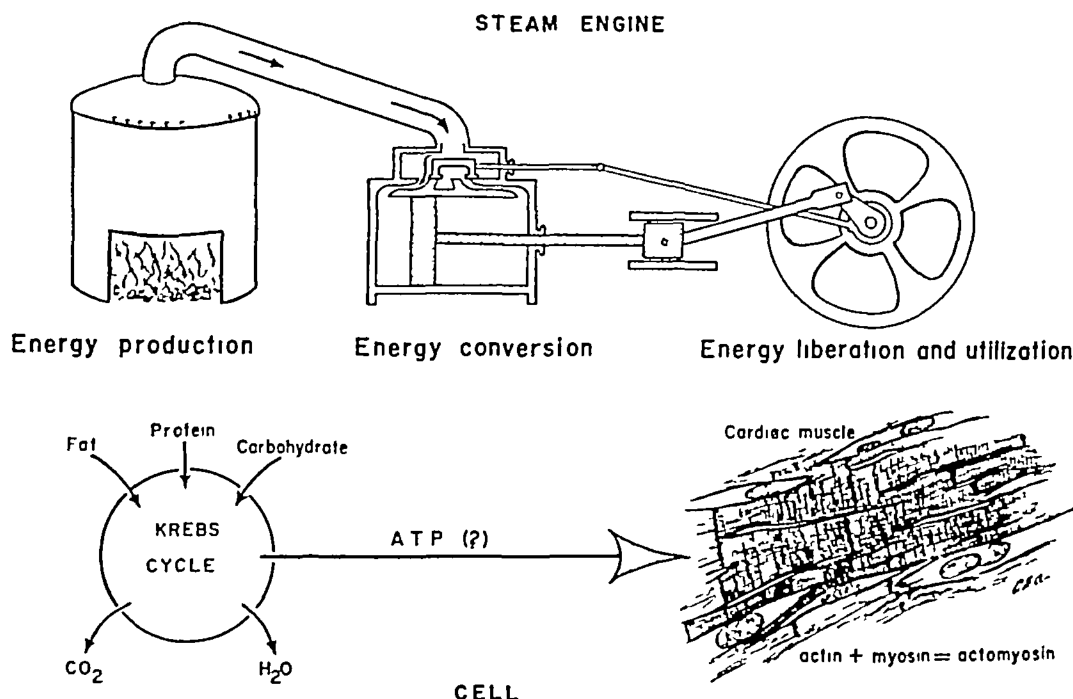


Fig 1 A comparison of energy-producing and energy-liberating processes in the mechanical system of the steam engine and in the heart Energy-producing processes in the steam engine are concerned with the production of steam through heat, in the cell, the main processes of energy production take place in the tricarboxylic acid cycle with the formation of high-energy phosphate

CATHETERIZATION OF THE CORONARY SINUS

I shall now discuss some of the applications of catheterization of the coronary sinus This technique has made it possible to study certain phases of coronary blood flow and myocardial metabolism Thus the catheter has opened up another field of physiologic research in man [15]

In discussing first the results obtained on the gaseous metabolism of the human heart, it could be shown that when the heart is performing a normal amount of work the arteriovenous oxygen difference across the coronary bed is almost maximal, thus the heart must rely entirely on changes in coronary blood flow to satisfy increased metabolic demands for oxygen [14] This is the case during exercise, when the load of the heart increases.[16] In normal individuals, the increase in coronary blood flow, leading to a rise in myocardial oxygen consumption, is accompanied by a proportionately greater increase in

the work of the heart; as a result, the myocardial efficiency, which represents the ratio of work of the heart to its oxygen consumption, is increased. In the failing heart, the rise in cardiac work is somewhat less than that in myocardial oxygen consumption, and the efficiency of the heart diminished.[16] Without going into detailed discussion of the various alterations in coronary blood flow which we observe under various conditions, I simply want to mention briefly that the coronary blood flow is markedly increased in anemia, and that it is likely that the amount of blood perfusing the coronary capillaries is increased in cardiac hypertrophy [14]

METABOLIC REQUIREMENTS OF THE HEART Intubation of the coronary sinus has made it possible to study certain phases of cardiac metabolism in man.[15] For clarity's sake, one can compare the heart to an engine, such as the steam engine (Fig 1) The energy-producing processes in the steam engine are concerned with the production of steam through heat, in the cell the main processes of energy production take place in the tricarboxylic acid cycle with the formation of high energy phosphate, the formation of energy-rich phosphate, as has been recently shown, takes place even beyond the substrate level, for example, in the hydrogen transfer system [17] The fuel which feeds this energy dynamo of the heart can be derived from carbohydrates, glucose, pyruvate and lactate, and from noncarbohydrate sources, ketones, amino acids and fatty acids As illustrated in Table 1, the sum of all

TABLE 1 RELATIVE CONTRIBUTION OF CARBOHYDRATES AND NONCARBOHYDRATES TO TOTAL MYOCARDIAL O_2 USAGE

Carbohydrate	%	*Noncarbohydrate*	%
Glucose	17 90	Fatty acids	67 0
Pyruvate	0.54	Amino acids	5 6
Lactate	16 46	Ketones	4.3
TOTAL	34 90%		76 9%

carbohydrates used by the heart muscle by no means covers the oxidative requirements of the heart. Only about 35 per cent of the energy requirements of the human heart are derived from carbohydrates, the rest must come from noncarbohydrate material, primarily fatty acids [15 18 19] It can be stated in general that in the human heart the uptake of carbohydrates by the heart is determined by their concentration in the arterial blood and that, as mentioned above, the total aerobic metabolism of glucose, lactate and pyruvate combined falls short of the total oxygen consumption of the heart; finally, cardiac work does not seem to influence the cardiac utilization of carbohydrates [25]

The statement has been made that the heart relies to a large extent on the usage of fatty acids [19] It has even been shown that the human heart can store fatty acids When the blood concentration of fatty acids is raised by the ingestion of a mixture of saturated and unsaturated fatty acids, the extraction of fat by the heart muscle rises considerably, indicating the storage of fatty acids [19] Amino acids are also removed by the heart, and their myocardial utilization appears to be stimulated by even small rises in their arterial con-

centration. Ketones are also used by the heart, it appears likely that the utilization of ketones by the heart is governed by their arterial concentration and by the quantity of carbohydrates available [19]

MYOCARDIAL METABOLISM. Of particular interest to us were the disturbances in myocardial metabolism. We have divided the disturbances in myocardial metabolism into those of energy production and those of energy utilization and liberation. As illustrated in Table 2, the disturbances in energy

TABLE 2 DISTURBANCES IN MYOCARDIAL METABOLISM

I Disturbances in energy production with reduced cardiac efficiency
  A Beriberi heart disease
  B Reduction in myocardial oxygen usage
    1 Hemorrhagic shock
    2 Coronary occlusion
    3 Ventricular fibrillation

II Disturbances in energy production without decreased cardiac efficiency
  A Diabetes

III Diminution in energy production and utilization with reduced cardiac efficiency
  A Hypothermia

IV. Disturbances in energy utilization with reduced cardiac efficiency
  A Congestive failure

production can be subdivided into those with normal and those with diminished cardiac performance. Obviously, beriberi heart disease is a condition in which a disturbance in energy production of the heart with diminished cardiac performance is present

The effect of reduction of myocardial oxygen usage or myocardial ischemia on myocardial metabolism is of particular interest. Conditions such as hemorrhagic shock, coronary occlusion and ventricular fibrillation have one thing in common· a reduction in coronary blood flow. We will see that the severity of the metabolic changes in the heart muscle depends to a large extent on the severity of the myocardial ischemia For example, in *hemorrhagic shock*, the reduction in coronary blood flow existing during the oligemic and normovolemic phases is accompanied by an increase in the concentration of pyruvate in coronary vein blood (Fig. 2) [20] Apparently pyruvate is not broken down to acetate to form acetyl-coenzyme A. This is probably the result of interference with the activity of the coenzyme cocarboxylase which has been shown to be destroyed into phosphate and thiamine in the presence of hypoxia [21] However, in hemorrhagic shock, the myocardial anoxia is not of sufficient severity to interfere with the breakdown of lactate by the heart muscle, as a result, the lactate balance of the myocardium remains positive [20]

The situation is different in *coronary occlusion* When coronary occlusion is produced in dogs by the injection of glass spheres of approximately 325 millimicrons diameter, a severe fall in blood pressure occurs[22] (Figs. 3, 4) The appearance of coronary shock is followed by disturbances in myocardial metabolism, such as negative myocardial pyruvate, glucose, lactate and inorganic phosphate balance (Fig 5) These changes are not always identical,

varying from animal to animal with the severity of the occlusion and myocardial ischemia produced. The question has arisen in the course of our work whether or not these metabolic derangements can be influenced by lowering the oxygen demands of the heart muscle. It is quite logical to assume that the metabolic effects of anoxia on the heart muscle vary with the ratio of oxygen

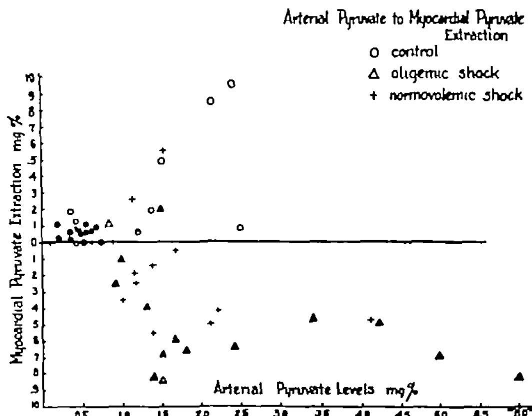


Fig 2. Illustrates the relationship of arterial concentration of pyruvate to myocardial pyruvate extraction in oligemic and normovolemic shock. The arterial concentration of pyruvate is elevated during both oligemic and normovolemic shock. Many of the points fall below the zero line, illustrating that the pyruvate concentration in coronary vein blood exceeds that in coronary arterial blood in many instances This pyruvate reversal" is present during both phases of shock. (From Journal of Clinical Investigation vol. 33, 1954)

demand to oxygen supply When the oxygen supply of the heart muscle is reduced in the presence of persistent high demands for oxygen by the heart muscle, the metabolic changes are expected to be severe. On the other hand, if the oxygen supply to the heart muscle is reduced together with the oxygen demand of the heart muscle, the metabolic effects of myocardial hypoxia should be less severe. Such studies are currently in progress and should be of considerable clinical interest.

Naturally, in *ventricular fibrillation* the myocardial anoxia is severe, thus metabolic changes within the heart muscle are expected to be profound This is illustrated in Fig 6 which shows that the concentrations of most metabolites in coronary vein blood exceeds that in arterial blood. In addition, the cell membrane has become extremely permeable to potassium and phosphate.

Mention was already made of the effect of hypothermia on the myocardial

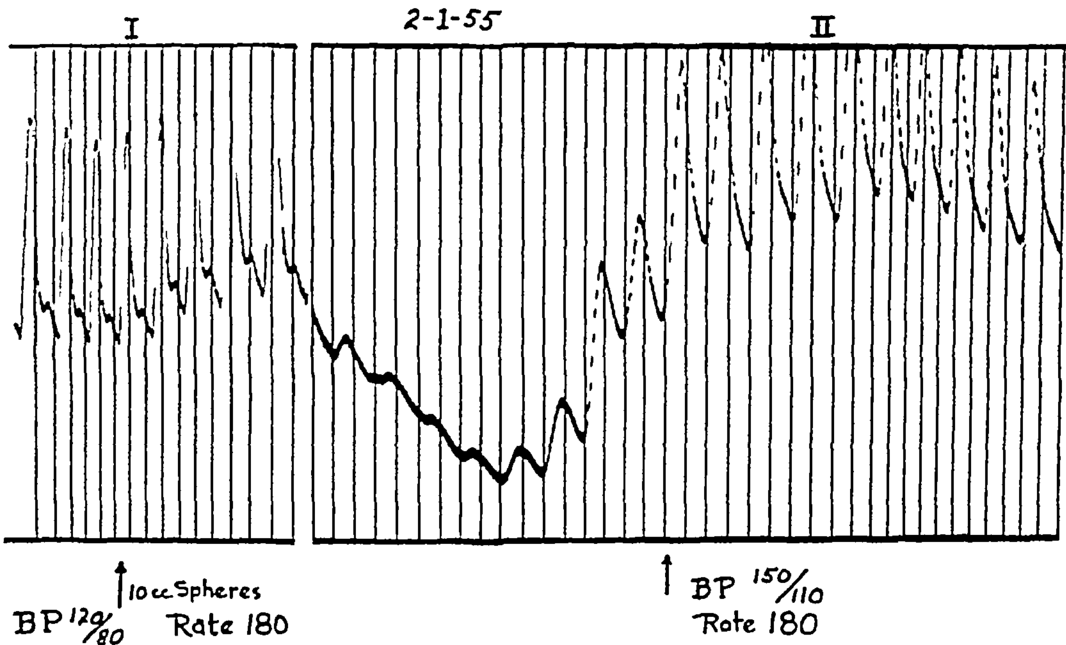


Fig 3

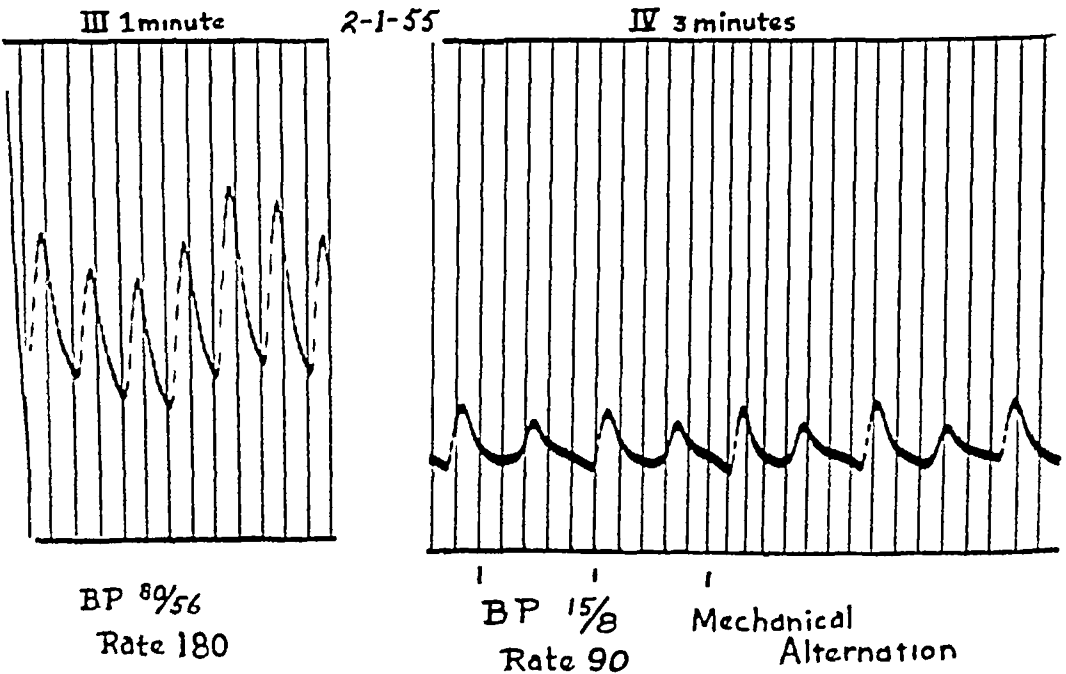


Fig 4

Figs 3 and 4 The fall in peripheral arterial pressure produced by the injection of glass spheres into the sinus of Valsalva It may be seen that the injection of glass spheres results in a marked drop in the peripheral arterial blood pressure from 120/80 mm to 15/8 mm Hg

metabolism. It has been shown that, although a reduction of body temperature to 28° C. leads to a marked diminution in coronary blood flow, metabolic effects which would be expected to result from this severe myocardial ischemia are absent. It is likely that metabolic changes do not occur because the ratio of myocardial oxygen demands to oxygen supply remains unchanged.[23]

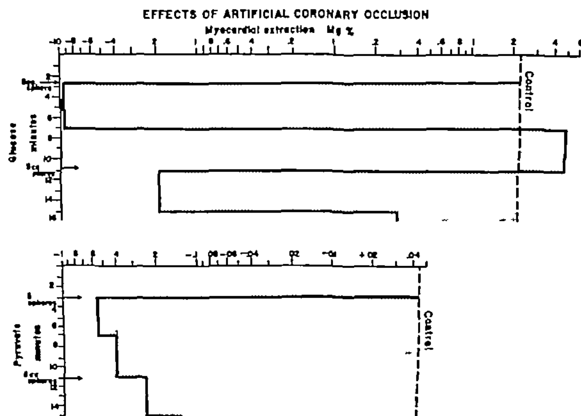


Fig. 5 The effects of coronary occlusion on the myocardial extraction of pyruvate and glucose. It may be seen that the injection of glass spheres into the sinus of Valsalva results in negative myocardial glucose and pyruvate balances

I will not dwell in detail on results obtained on patients with diabetes and myocardial failure, except to state that our studies have shown that in diabetes, the metabolic defects within the heart muscle extend not only to myocardial utilization of carbohydrates but also to fatty acids and amino acids as well [24]

In myocardial failure, subsequent to valvular disease or hypertensive cardiovascular disease, the extraction of all substrates by the heart muscle appears to be within normal limits This suggests that the disturbance in congestive failure is located in the structures concerned with energy liberation, the contractile proteins Although it is likely that the pathways leading to myocardial failure can have a variety of origins, all the roads finally join and failure of energy liberation is the ultimate result.

I have attempted to present, in this brief span of time, some of the advances which catheterization of the heart has attained in the field of cardiac physiology I have, purposely, paid no attention to the value of catheterization in the diagnosis of congenital or acquired cardiac defects These facets of this

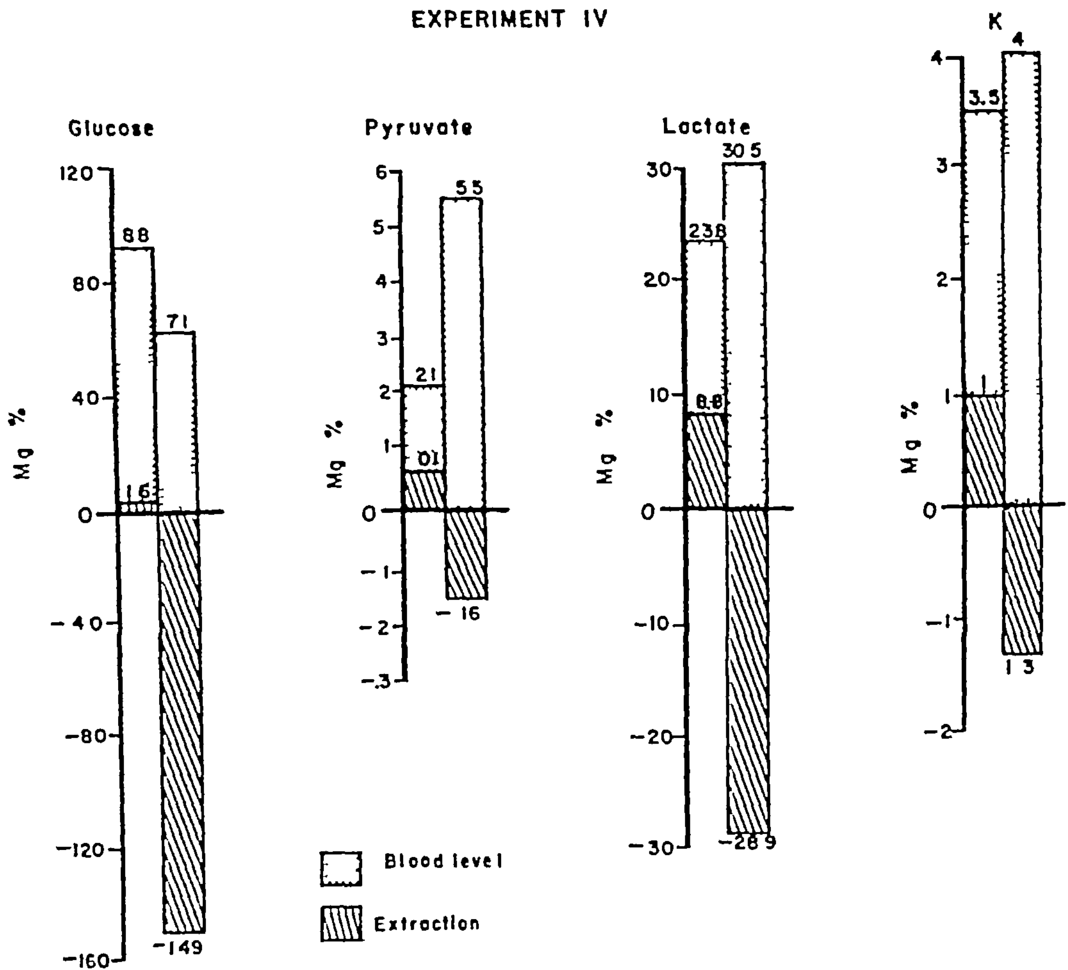


Fig 6 Shows the changes in the myocardial metabolism following ventricul fibrillation The concentration of glucose, pyruvate, lactate and potassium in coronar vein blood exceeds that in arterial blood following the onset of ventricular fibrillation

technique are known to most of you, and will be discussed in further portion of this Symposium Undoubtedly, the greatest value derived from cardia catheterization has been gained through studies which have dealt with trul fundamental aspects of circulation.

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Question Have you had any theory as to the frequency of ventricular fibrillation under hypothermia?
Answer There will be a panel on this subject later in this Sympo-

sium, and I should refer most of the questions to the members of that panel.

I would like to mention the work of Dr. Henry Swan, in which he has indicated some disturbances in cholinesterase mechanism in fibrillation, but I would like also to mention some work which has been dor in Birmingham by Dr. Sterling Edwards which indicates very clear that one can cut down on the incidence of ventricular fibrillation t increasing the coronary blood flow during that state.

In other words, if there is a certain increased amount of blood goin through the coronary circulation in the presence of hypothermia, tl incidence of fibrillation is very markedly decreased This is an inte esting finding in the light of our almost contradictory evidence th metabolic changes are not present in hypothermia despite the ver marked reduction in the coronary blood flow.

Question: I would like to ask Dr. Bing if he has any information o changes in the ratios of carbohydrate utilization and fatty produc utilization under hypothermia with inflow occlusion and therefor anoxia.

Answer: The answer is apparently no. We have studied many patient in hypothermia, with no change in carbohydrate metabolism, and in th few in which we studied amino acids, fatty acids and ketones, there wa no change.

Question: Does use of a catheter with manometer at tip add anything In other words, are usual technical methods adequate?

Answer: The Mayo group has done work in this field, and, as yo probably all know, many years ago a little manometer was devise which goes to the tip of the catheter. Unfortunately we have ha no experience with this instrument but the recorded pressure curve from the Mayo Clinic are very encouraging indeed.

Since this is a forum in which new data can be presented, I will simpl say that a new type of catheter looks very promising. It is manufacture by the Du Pont Company All of those who do catheterization kno how many times it is impossible to introduce the catheter into the pul monary artery A catheter that can be bent in any direction by mean of nylon threads is being devised by Du Pont, and I think it should b available very shortly.

Question: Has the speaker had embolic complications from catheterization in the presence of right to left shunts? Have catheters been treated with silicone preparation to prevent clot formation in catheters?

Answer: First, I will answer the question about the embolic complications in right to left shunt.

In the early days of catheterization, in 1946, we did have air embolism —at least what looked like it—in certain situations. In four successive catheterizations, there were cerebral symptoms This experience has not

been repeated. This type of accident is more likely to occur in conditions in which the aorta overrides There have been pulmonary emboli described in those conditions in which the pulmonary capillary pressure is measured and in which the catheter is put in position as a wedge catheter for longer than a minute or so Dr Dexter's experience has been similar, and we had one patient who developed a pulmonary embolus following maintenance of the catheter in the wedge position.

Concerning the silicone, I do not think it is necessary We always drop some diluted heparin through the catheter, and clots seldom form around the catheter

I would like to say that we find it most advantageous to introduce a stylet into the catheter, because then one can boil the catheter and avoid the complications of chills, which one so very frequently observes when the catheter is not boiled but is simply sterilized in a solution of Zephiran or something of that sort.

THE ELECTROCARDIOGRAM IN CONGENITAL CARDIOVASCULAR DISEASE

ROBERT F. ZIEGLER (*Detroit*)

While much work remains to be done to establish more adequate ai accurate diagnostic criteria, it is now well known that one of the most si nificant contributions of electrocardiography to the study of patients wi congenital cardiovascular defects is evidence of single or combined chamb enlargement It is perhaps not quite so well known that the electrocardiogra provides information which is frequently superior to that obtained radiolog cally in determining not only the presence but also the degree of ventricul hypertrophy One of the newest and most significant contributions, howeve is physiologic rather than anatomic, and provides evidence of the altere hemodynamics underlying the type of enlargement observed. It is not clain ing too much for the electrocardiogram to state that from it one can estima with reasonable accuracy right ventricular work and therefore approxima mean right ventricular pressure, and make a reasonable inference as to wheth the increased work and enlargement of the right or left ventricle are due t normal or even decreased output against increased resistance or to increase output with normal resistance The factors involved in this latter differenti tion correspond to the systolic and diastolic overloading patterns respectivel of Cabrera and Sodi-Pallares.[1,2,3]

It would be well at this point to define the physiologic and electrocardic graphic terms to be used Excluding primary myocardial disease, arrhyth mias, and the like, most of the congenital cardiovascular defects can b classified according to the presence of (1) obstruction or increased resistanc to blood flow, such as pulmonary stenosis, pulmonary endarteritis, coarctatio of the aorta, and (2) a communication and shunt between the systemic an pulmonary circulations, such as interatrial and interventricular septal defect and patent ductus arteriosus, or (3) various combinations of these two basi types Each uncomplicated situation will be associated with enlargement o specific cardiac chambers· the first, of those on the side of the increase resistance to blood flow, the second, on the side receiving the shunt. Ven tricular enlargement in the first situation is that of normal or even decrease cardiac output with elevated systolic and mean intraventricular pressures, an

corresponds to the systolic overload pattern of Cabrera and Sodi-Pallares Ventricular enlargement in the second is that of increased volume of cardiac output against normal peripheral resistance with normal or only slightly increased intracardiac pressures, and corresponds to the diastolic overloading pattern of these investigators Combinations of these two basic types of defects obviously but significantly will produce combined right and left ventricular enlargement.

Cabrera and Sodi-Pallares define their systolic and diastolic overloading patterns for the right and left ventricles as follows:

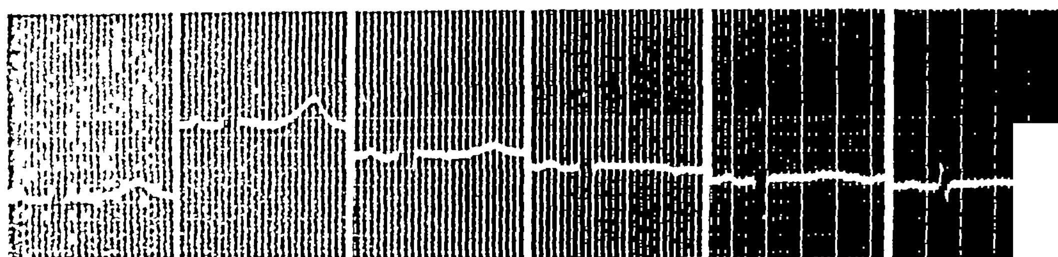
Systolic
- Right ventricle
  - Increased amplitude of R } right precordial leads
  - Negative symmetrical T } right precordial leads
- Left ventricle
  - Negative ST segment and inverted T waves in left precordial leads

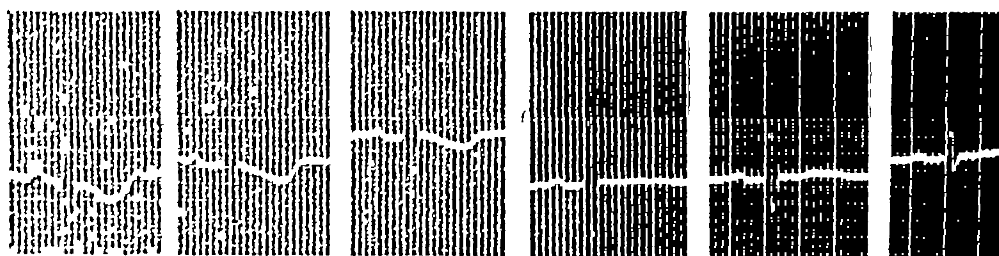
Diastolic
- Right ventricle
  - Incomplete or complete right bundle branch block
- Left ventricle
  - Tall R } left precordial leads
  - Late activation time } left precordial leads
  - Tall upright and symmetrically peaked T } left precordial leads

The foregoing hemodynamic and electrocardiographic classification has proved to be of inestimable clinical value. However, sufficient numbers of factors are involved in the interpretation of the various electrocardiographic deflections and in the various leads that the determination of specific diagnostic criteria must first be submitted to careful and critical statistical analysis Among the most important variable factors are those due to age—and not only the normally changing ratio of right and left ventricular size but also the age changes which occur in the natural progression of each type of congenital cardiovascular defect beginning with the pattern of the fetal circulation. Assuming that the precordial electrocardiogram provides accurate evidence of the relative size of the right and left ventricles, whether normal or abnormal, then a detailed statistical analysis of normal measurements is necessary for each important age group before the significance of variations from normal can be stated. Since early infancy is one of the most critical periods with respect to management and important for our knowledge of the natural history of various defects, and since this period, until very recently, has been almost completely unstudied, we have selected from among infants several problems illustrative of the clinical value of the electrocardiogram in terms of the concepts already defined.

One of the most pertinent electrocardiographic observations in normal infants is the frequency of upright T waves in right precordial leads during the first 24 hours after birth and the progressively increasing negativity of this deflection immediately thereafter (Fig 1) Several explanations have been

offered, among which the most attractive is the initially high pulmonary artery and right ventricular pressures which decrease with expansion of the lungs and decreased pulmonary arteriolar resistance (Figs. 2 and 3). Starting





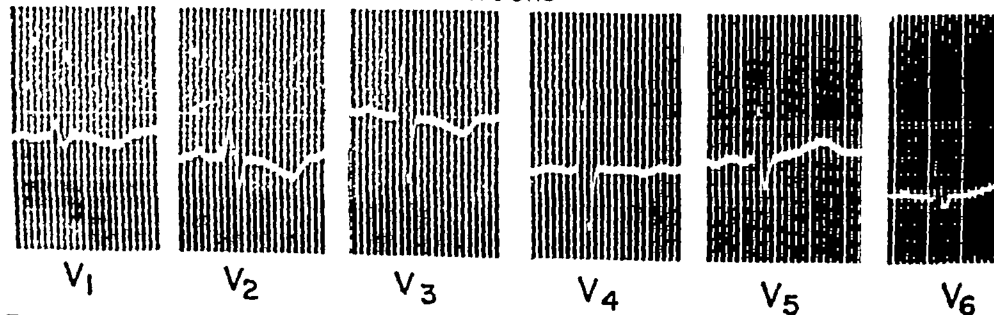


FIG 1. Unipolar precordial leads V_1 through V_6 in a normal infant The u T waves in leads from the right side of the precordium are characteristic of th 24 hours of postnatal life only, and after this period are highly suggestive pathognomonic of pathologic right ventricular hypertrophy. For further d see text

from this observation, and with the assumption that the presence and d of right ventricular hypertrophy within any particular age group sl parallel increased right ventricular work, it was found that there is an app relationship between the incidence of positive T waves in lead V_1 an degree of elevation of right ventricular mean pressure. A more detailed ysis of the T wave pattern according to both right ventricular mean pre and the QRS pattern in right precordial leads reveals the following (Fi

1 There is a reciprocal relationship between the net area of QRS and that at all levels of right ventricular pressure (Fig 5).

2 Within any given QRS pattern there is a direct relationship between the inci of positive T waves and the degree of elevation of right ventricular mean pressu

Fortunately the relationship between QRS and T is such that the diagnostic value of an upright T in lead V_1 is greatest when QRS is normal (Table 1), and this represents one of the earliest recognizable patterns of systolic overloading or pressure-work hypertrophy of the right ventricle (Fig 6) With

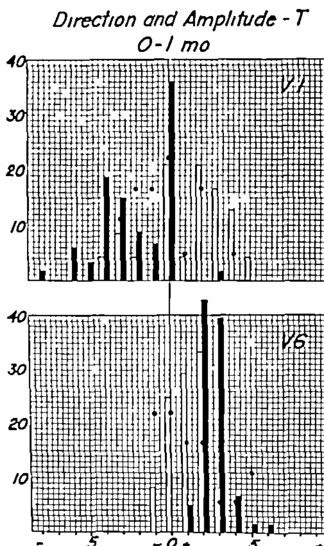


Fig 2. In this illustration the solid columns represent the normal direction and amplitude of precordial T waves *after* the first day of postnatal life The open columns represent this normal distribution during the first day only The solid circles represent the distribution of T wave measurements in cases with known right ventricular hypertrophy

greater degrees of this type of right ventricular enlargement, with or without the addition of so-called "diastolic overloading, the frequent superimposition of some degree of right bundle branch block will convert the ventricular deflections in right precordial leads into the typical form previously described (Fig 7)—large amplitude of R with variable notching, late onset of the intrinsicoid deflection, small or absent S and inverted T It is also likely that this latter pattern represents the end stage of marked right ventricular hypertrophy due to increased output with normal or only slightly to moderately increased peripheral resistance (Fig 8), so that at this point the two types

may be practically indistinguishable. Lesser degrees of pure diastolic overloading and right ventricular enlargement, with incomplete or even complete right bundle branch block, are difficult to differentiate from uncomplicated right bundle branch block in the normal heart, especially during infancy when

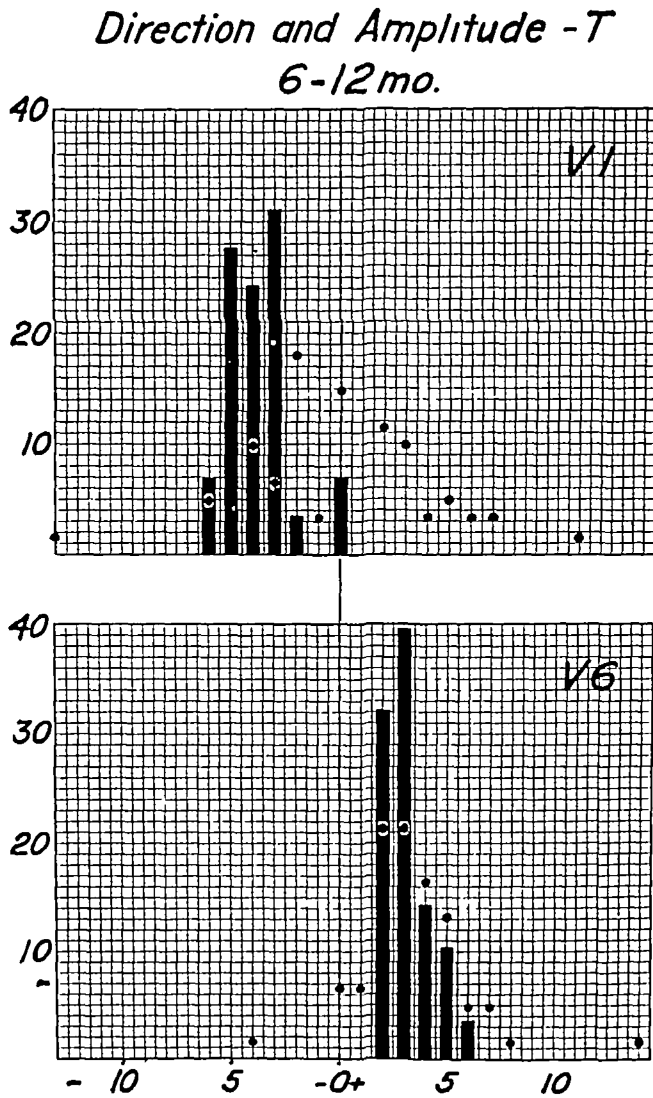


Fig 3 In this illustration the solid columns represent normal and the solid circles abnormal distribution (right ventricular hypertrophy) of T wave measurements in unipolar precordial leads In this as in Fig 2, note the *positivity* of T waves in leads from the right side of the precordium in right ventricular hypertrophy

the relative size of the right ventricle is normally greater than in older age groups. This latter constitutes a problem of great clinical importance, for which time does not permit discussion here

Even though the concept of diastolic and systolic overloading of the left ventricle appears to be valid, at least under certain clinical circumstances, there is also evidence to suggest that these terms could be applied respectively to lesser and greater degrees of simple left ventricular enlargement. The best example of pure diastolic overloading would be that of tricuspid atresia with total venous return reaching the left ventricle (Fig 9*A*), plus the additional

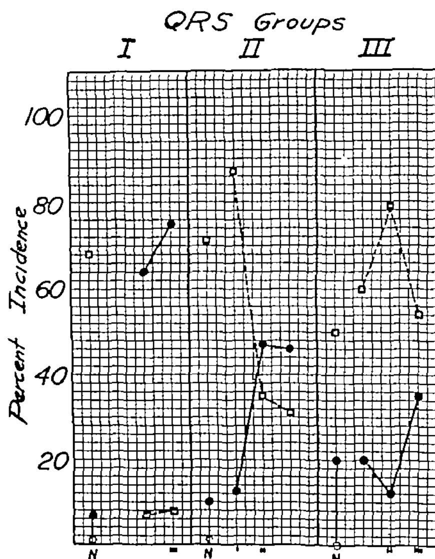


Fig 4. This figure represents the percent incidence of upright (solid circles) and inverted (open squares) T waves in precordial lead V_1 at different levels of mean right ventricular pressure (I slight II moderate and III marked elevation) and with different configurations of QRS (I amplitude of R = 60% of total amplitude of RS, the R/S ratio = 1 or less, total area of R + S = approximately zero, II amplitude of R = 60–85% of total amplitude of RS, the R/S ratio = greater than 1, total area of R + S = moderate positivity III, amplitude of R = 85–100% of total amplitude of RS the R/S ratio and total area of R + S approaches maximum positivity) It is to be noted that as the area or amplitude of R with respect to that of S increases there is a decreasing incidence of positive T waves in lead V_1 at all levels of mean right ventricular pressure. However within each QRS group there is an increasing incidence of upright T waves in lead V_1 with increasing mean right ventricular pressure.

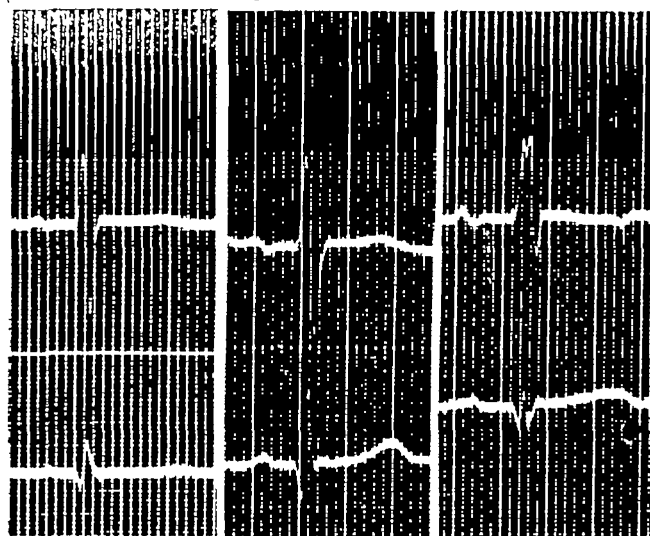


Fig 5 Simultaneous leads V_1 (above) and V_6 (below) in the same child with a proved tetralogy of Fallot, at various ages There is increasing right ventricular hypertrophy, the first evidence for which is the upright T in lead V_1 at the age of 7 weeks, at which time the configuration of QRS in this lead is entirely normal As the total positive area of QRS increases that of T reciprocally decreases, as observed at the age of 1 year

TABLE 1

	I	II	III
Ampl of single-peaked R = % RS, lead V_1 Group distribution	– 60 22 5	60–85 33 3	85–100 44 2
Positive Diagnosis of Right Ventricular Hypertrophy			
Activation time T wave pattern	19 1 76 3	63 3 46 7	85 2 25 8

The QRS groups designated I, II and III are the same as those in Fig 4 It is to noted that the diagnostic value of the T wave pattern is reciprocally related to that of QRS

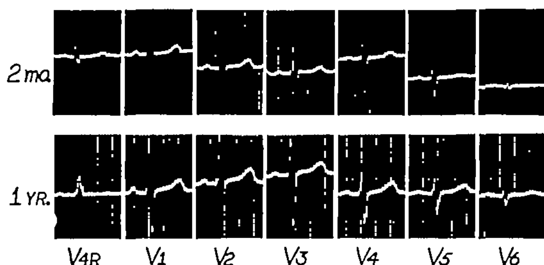


Fig 6 Typical precordial electrocardiogram in uncomplicated tetralogy of Fallot especially during infancy Note the normal configuration of QRS with upright T waves in lead V_1

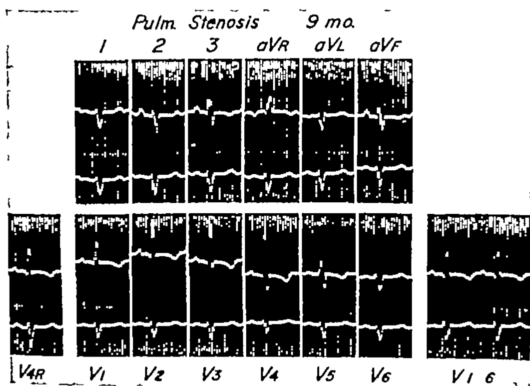


Fig. 7 An electrocardiogram representative of a greater degree of pressure-work hypertrophy than illustrated in Fig. 6 Note the initial Q, the larger positive amplitude and area of R, and inverted T in leads from the right side of the precordium.

diastolic load imposed by the creation of an artificial patent ductus. According to the aforementioned definitions one would expect the R and T deflections in left precordial leads to become more positive. Instead, it is more usual to observe ST segment depression and T wave inversion (Fig. 9*B*). This latter is also a perfectly typical pattern with a physiologically large and uncomplicated patent ductus arteriosus (Fig. 10), as well as that to which the diastolic overloading pattern may be easily converted by the administration of digitalis. It would seem fair to conclude that in the case of either isolated right or left ventricular hypertrophy the differentiation between the two types of enlarge-

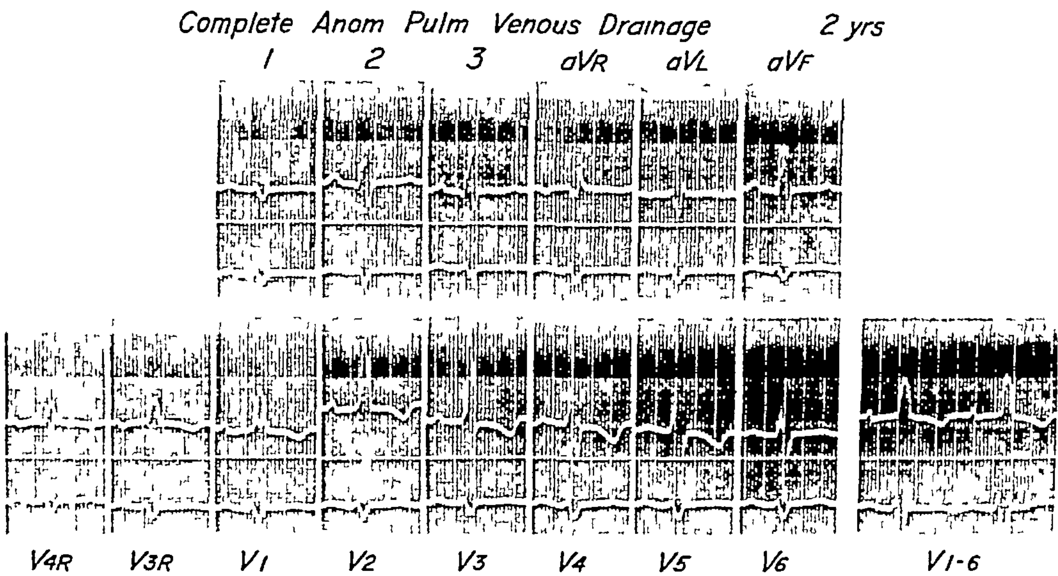


Fig. 8. An electrocardiogram in a clinical situation characterized physiologically by marked diastolic overloading of the right ventricle Note the similarity between this and the pattern illustrated in Fig 7

ment is most practicable and clinically useful when this enlargement is only moderate, and when enlargement of either ventricle is marked, other factors such as combinations of right and left ventricular hypertrophy become of much greater importance.

It might first be stated that combined enlargement of both right and left ventricles does not "balance" the electrocardiogram back to normal, which implies that positive and specific criteria should be in evidence for each separate entity. Again, time does not permit a complete discussion of this problem, but in outline several important diagnostic patterns should be mentioned

1. Pure diastolic overloading of both right and left ventricles, though uncommon, does exist in several combinations of defects. One example which would satisfy the physiologic criteria is patent ductus arteriosus with pulmonary valvular insufficiency (Fig 11). The electrocardiographic pattern in this situation consists of the typical high R with symmetrically peaked upright T waves in left precordial leads and uncomplicated right bundle branch block in leads from the right side of the precordium.

2. Systolic overloading of the right with diastolic overloading of the left

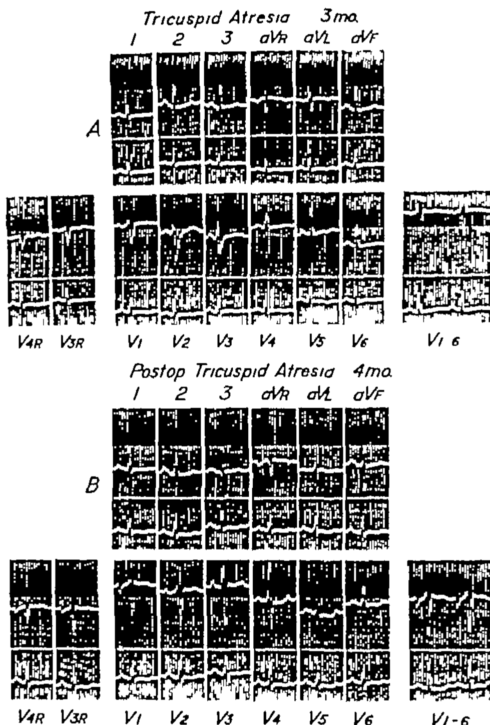


Fig 9 *A* A typical electrocardiogram in tricuspid atresia. *B* An electrocardiogram taken one month after the creation of an artificial patent ductus arteriosus (Potts operation) in the same patient as illustrated in Fig. 9*A* Note the inversion of T waves in left precordial leads, also the P wave configuration which is characteristic of left atrial enlargement.

Patent ductus 4 mo.

1 2 3 aVR aVL aVF

V4R V3R V1 V2 V3 V4 V5 V6

FIG 10. An electrocardiogram in an infant with uncomplicated patent ductus arteriosus and receiving digitalis There was combined right and left ventricular enlargement anatomically The upright T waves in right precordial leads probably reflect the elevated pulmonary artery and right ventricular pressures present in this case. Note also the absence of the so-called typical diastolic overloading pattern of the left ventricle

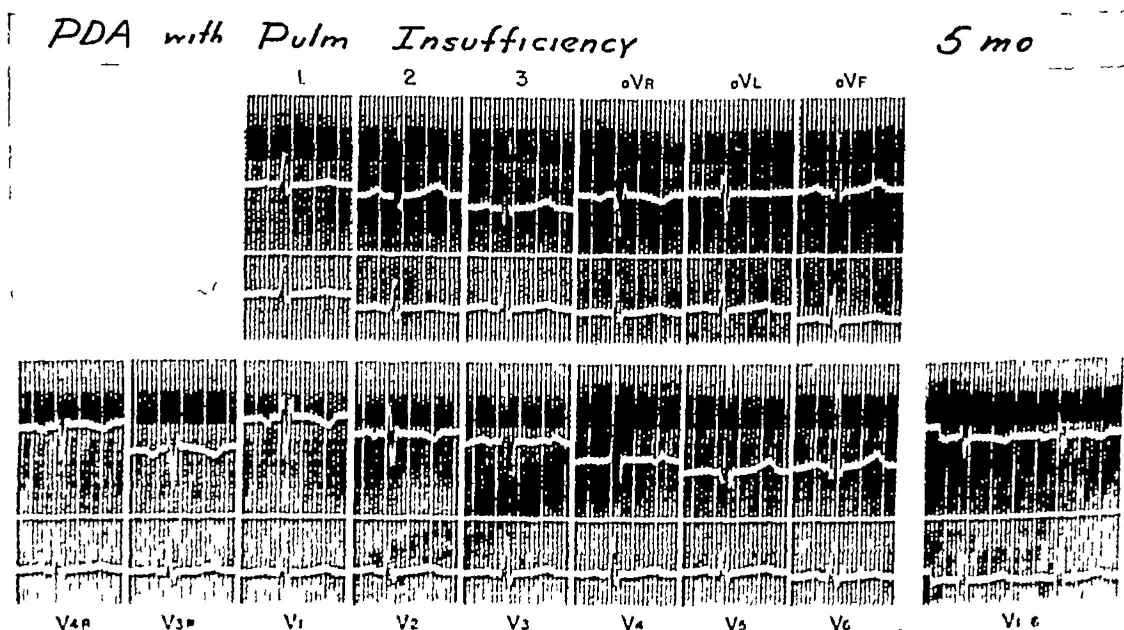


FIG 11. The electrocardiogram in patent ductus arteriosus with pulmonary valvular insufficiency proved by cardiac catheterization and surgery. There is left ventricular hypertrophy and right bundle branch block

ventricle. This pattern, characterized by T wave positivity in leads from both sides of the precordium but with larger amplitude R in V_6 than would be anticipated in uncomplicated right ventricular hypertrophy, is observed in severe pulmonary valvular stenosis with an intact interventricular septum and a relatively large right-to-left interatrial shunt (Fig 12)

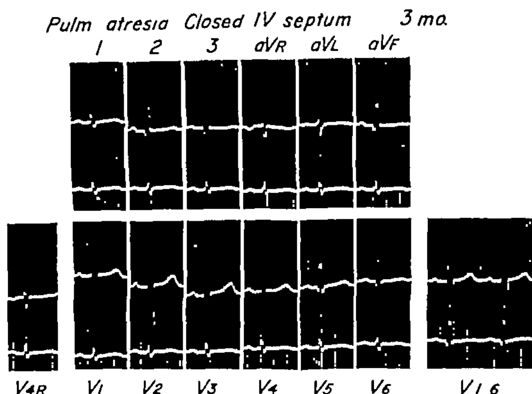


Fig. 12. The electrocardiogram in a clinical condition characterized physiologically by systolic overloading of the right and diastolic overloading of the left ventricle There is combined right and left ventricular hypertrophy, with upright T waves in right precordial leads diagnostic of the former and QRS configuration and measurements in left precordial leads diagnostic of the latter

3 Systolic overloading of the left ventricle with diastolic overloading of the right. An example of this situation, by physiologic definition, would be aortic coarctation with a distal patent ductus arteriosus (Fig 13) In this situation age changes are exceedingly important. However, one might expect right bundle branch block to dominate the right precordial lead pattern with late activation time in leads from the left side of the precordium. T wave changes in left precordial leads might be masked by the transmitted effects of the bundle branch block pattern.

4 Systolic overloading of both right and left ventricles, except for combined aortic and pulmonary stenosis, is likely to be a physiologically mixed situation involving left-sided obstruction as coarctation of the aorta together with a left-to-right shunt (interventricular septal defect or proximal patent ductus) and increased pulmonary arteriolar resistance (Fig. 14) The electrocardiographic findings may vary also, and include mixtures of QRS and T wave alterations on both sides of the precordium.

SUMMARY

1. The clinical value of the electrocardiogram in congenital cardiovascular disease depends only partly on the accuracy with which it provides evidence of single or combined cardiac chamber enlargement. Another extremely important contribution is the evidence it provides of significant alterations in hemodynamics, with or without other evidence of physiologic "strain" or anatomic enlargement.

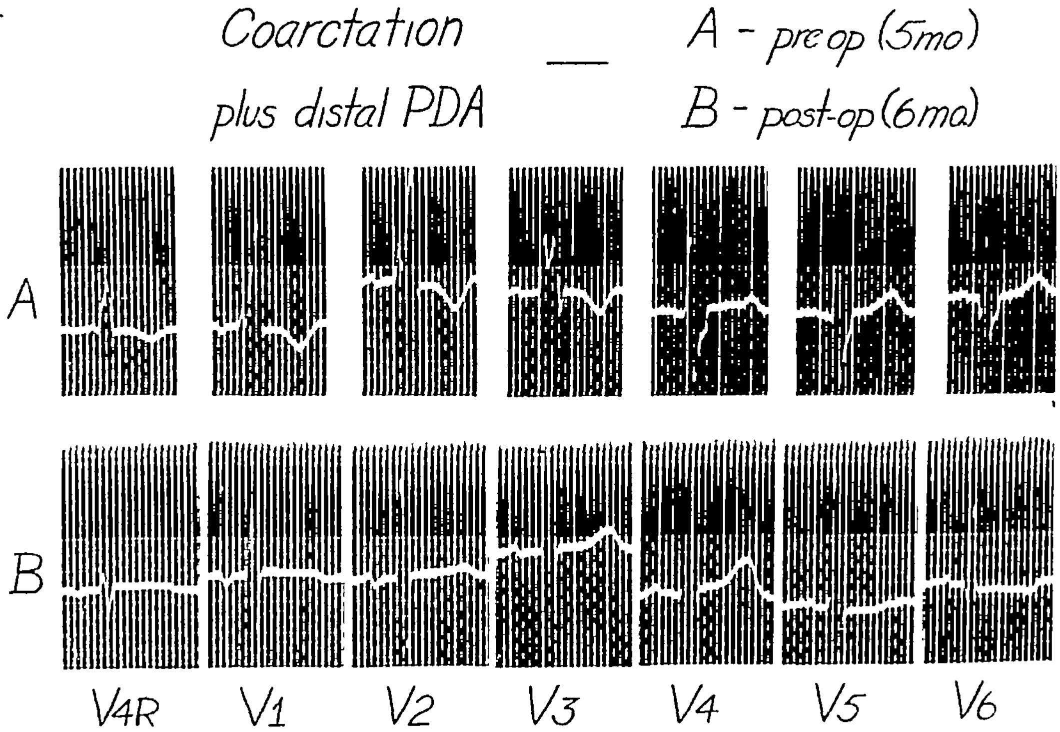


Fig 13 Pre- and postoperative precordial electrocardiograms in coarctation of the aorta with a distal patent ductus arteriosus Note the change in pattern with reduction of right ventricular mean pressure

2 It is to be emphasized that even the unipolar precordial leads are subject to a number of variable factors and require careful and critical analysis before permitting final clinical conclusions to be made.

3. It was stated introductorily that this paper would concern itself primarily with electrocardiograms in infants. Most if not all of the basic principles discussed, however, apply equally to all age groups Care must again be used in considering the important changes due to age for the determination of actual numerical criteria The changes to be considered include not only those incident to normal growth and development but also those which appertain to the natural history of each cardiovascular defect, beginning with the pattern of the fetal circulation

4 Several of the most important clinical and physiologic problems, with representative electrocardiographic patterns, are discussed.

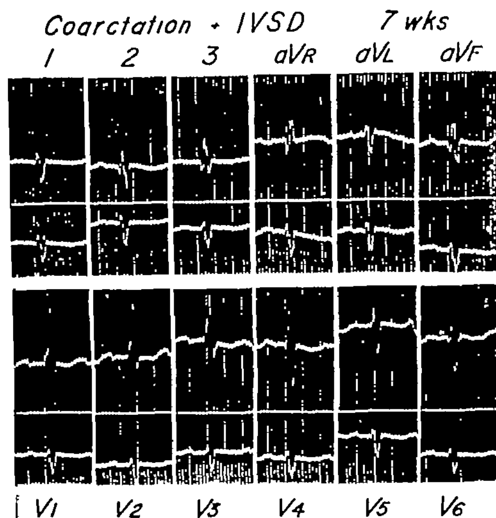


Fig. 14 The electrocardiogram in coarctation of the aorta plus an interventricular septal defect, with marked pulmonary artery and right ventricular hypertension. There is QRS evidence of right and T wave evidence of left ventricular hypertrophy, one of the types of patterns characteristic of combined ventricular enlargement.

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Question Since the height and symmetry of the T waves in V_5 and V_6 and V_7 vary considerably in normal children, what are your criteria for the diagnosis of diastolic overloading of the left ventricle?

Answer As far as systolic overloading of the right ventricle is concerned, I suggested that increased positivity of the T wave in right precordial leads is the first and most important sign of minimal and then increasing degrees of this abnormality

The second change in succession would be increased area or increased positivity of the QRS deflections in leads from the right side of the precordium. I should also repeat that as the area of the QRS becomes more positive, the area of T reciprocally becomes more negative, still with systolic overloading of the right ventricle.

Systolic overloading of the left ventricle has been characterized by increased negativity of the final ventricular deflections, the ST segment and T wave. I would like to change that only to the extent that there is sufficient evidence, I think, that this represents a marked degree of left ventricular enlargement, whether it begins with systolic or diastolic overloading.

Diastolic overloading of the right ventricle has been quite properly described as characterized by incomplete or complete right bundle branch block, whatever other changes might be superimposed.

Diastolic overloading of the left ventricle has been described as characterized by increased positivity of both the initial and final ventricular deflections in leads from the left side of the precordium. Here we get into trouble with comparing the numerical data with the normal for that age. We also must consider the numerical measurements from the opposite side of the precordium.

We have not as yet completed studies which are in progress for the actual determination of numbers which would permit this diagnosis. As a matter of fact, one of the things we are going to have to do (and are in the process of doing) is to relate the total area under QRS to the total area under T in both leads from the right and left sides of the precordium, and see what numerical answers we get.

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DISCUSSION

F. D. Johnston (*Ann Arbor, Michigan*)

There is one word of caution that perhaps might be mentioned. I do not agree 100 per cent on the interpretation of the T wave changes in quite as definite form as Dr. Ziegler suggested After all, the T waves are easily altered by many factors I believe that the implication that left ventricular hypertrophy is present because of inverted T waves over in leads from the left side of the precordium may be something that should be taken with a little bit of caution, at least.

Dr. Ziegler

Dr. Johnston is perfectly right in commenting on the variability of changes which we here in a brief period interpreted specifically. I also tried to allude to that. It is certainly worthy of reemphasis that there are many other non-cardiac factors which may produce some of these electrocardiographic changes that have been described. For example, some of the electrolyte changes could

have produced electrocardiograms which we could have shown here as perfectly typical of diastolic or systolic overloading

Obviously, it is extremely important to correlate the electrocardiographic findings with all of the other available clinical information.

RECENT DEVELOPMENTS IN ANGIOCARDIOGRAPHY

JOHN LIND (*Stockholm*), CARL WEGELIUS (*Stockholm*) AND IB BOESEN (*Stockholm*)

One of the main problems, in discussing the present status of angiocardiography, is to delineate the extent of its practical utilization. This relatively new technique has already become established as a routine examination for the investigation of congenital heart malformations. Because of continued progress in the technical field, improvements in performance and recording, as well as interpretation, can be expected. Since the procedure involves considerable risk to the patient and expense and time on the part of the investigators, a review of all available technical improvements is in order. These improvements have not only broadened the scope of investigation but have also made possible a more precise interpretation of the deranged anatomy. For the sake of clarity, we shall discuss the various problems individually, although they are naturally dependent on one another.

RECORDING FACILITIES

The x-ray industry is now able to furnish us with devices of fairly good quality There are two principal types of recording apparatuses. (1) the direct type, recording in normal size, and (2) the indirect type, recording by photofluorography on film of diminished size. The Schonander single film changer (Fig. 1) and the Elema roll film device (Fig. 2) are examples of the direct type. The Odelca in 70 mm film size (Fig. 3) and the Philips device with image intensification in 35 mm. film size are examples of the indirect type (Figs. 4–6)

BIPLANE RECORDING

This is considered valuable for the following reasons:

1 Three-dimensional interpretation of the cardiac chambers and the heart as a whole

2 Interpretation of overlapping of heart chambers in one of the projections

3 Avoidance of the risk associated with repeated injections of the contrast medium

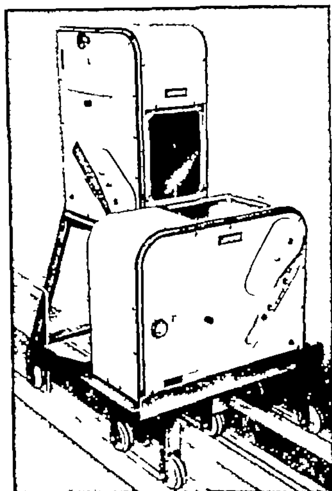


Fig 1 The Schönander AOT film changer for rapid serial radiography with cut film in standard sizes 10 x 12 inches and 14 x 14 inches Exposure frequency 1–6/sec.

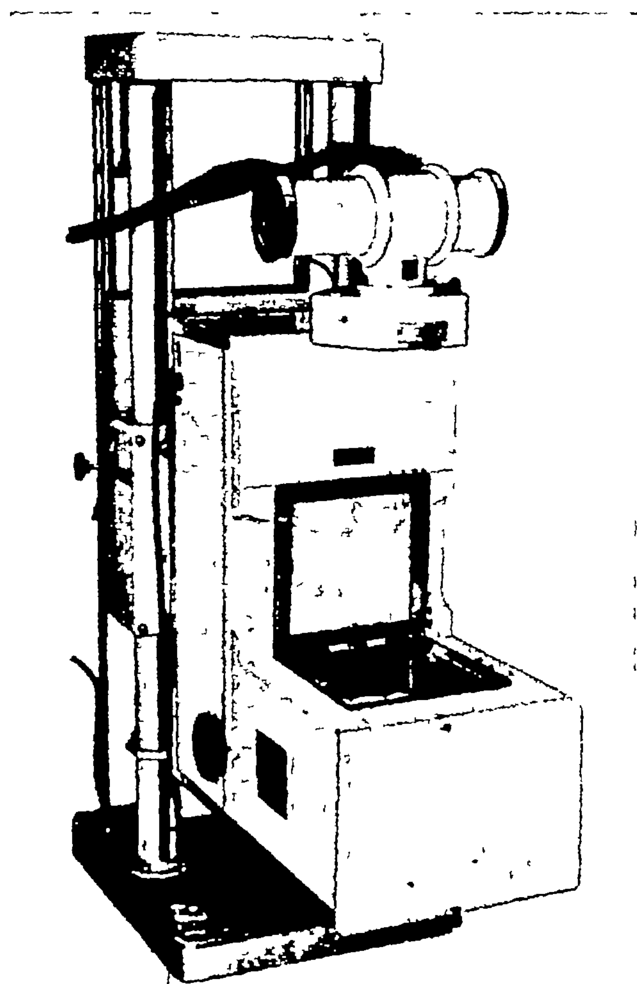


Fig 2 Elema roll film device for rapid serial radiography, 30 cm Exposure frequency 1–8/sec.

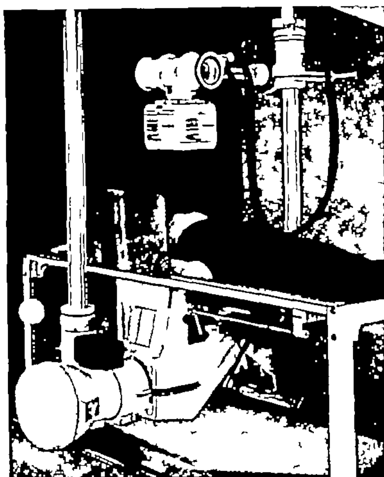


Fig. 3 Odelca photofluorographic x ray camera for serial radiography Film size 70 mm. Exposure frequency 1–6/sec.

EXPOSURE FREQUENCY

A high rate of exposure is necessary to study the dynamics of the heart. A minimum frequency of 3 exposures per heart cycle is sufficient for filming the extremes of the systolic and diastolic heart phases, as well as the intermediate stages. Both direct and indirect types of devices fulfill these demands with normal heart rates. The image intensifier with exposure frequencies of up to 50/sec. permits additional possibilities for the study of cardiac dynamics.

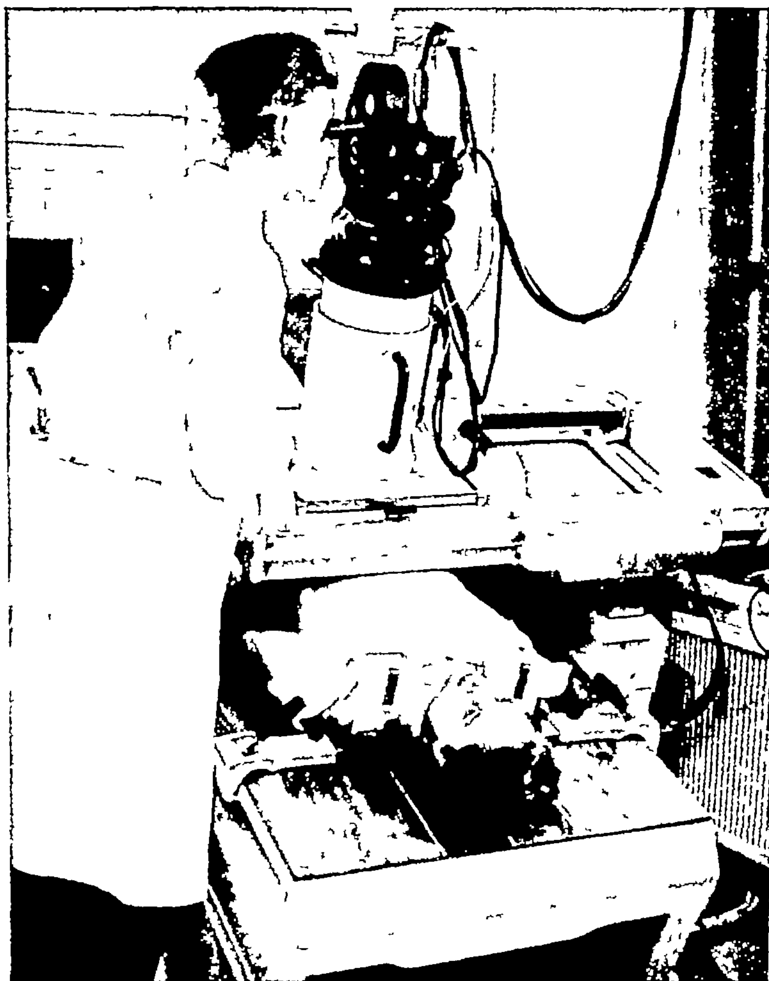


Fig 4 Philips image intensifier with Arriflex cine-camera fixed to symmetric couch in horizontal position Norrtulls Hospital, Stockholm. Film size 35 mm Maximum exposure frequency 50/sec

EXPOSURE DATA

With regard to the rapid morphologic changes of the heart due to contractions and other intrinsic movements, the output of the apparatus must be great enough to use exposure times of not longer than 0 02 sec in order to avoid blurring of the pictures This demands a high milliamperage—for adults 500 to 600 ma. for each tube at the conventional 100 kv. At increased kilovoltage of 120 to 150, the milliamperage can be reduced to ½ or ¼. The image intensifier can be operated with much lower output (2 to 5%) than the direct types and with exposure times of about 0 01 sec at a maximal

TABLE 1. EXPOSURES REQUIRED FOR DIFFERENT TYPES OF RECORDING

A-P angiocardiogram of middle-sized adult at 1 m focal distance

Single exposure requires for:	kv.	80	100	125
Photofluorography, mirror image	mAs	100	25	6
Direct radiography		30	8	2
Image intensification recording		0 8	0 3	0 1
(Exclusive exposure values, total radiation doubled because of uninterrupted x-ray tube discharges)				

exposure rate of 50/sec. Photofluorography with the Odelca device requires somewhat more milliamperage than direct radiography (Table 1).

IRRADIATION DOSAGE

The dosage of irradiation is directly related to the exposure data. The problem of irradiation and its dangers must be taken into consideration. The upper limit of safe irradiation for angiocardiography has not been established but should be kept as low as possible. A diagnostic irradiation of approximately 10 to 50 r, which occurs in other complicated radiographic examinations, should not be exceeded if possible. All available tools should be used to decrease the absorbed rays. Among them, the high kilovoltage technique is the most valuable, however, more sensitive films, intensifying screens and developing procedures may also, in effect, decrease the total exposure. The image intensifier naturally offers the optimal conditions for minimal irradiation (Table 2). When cardiac catheterization precedes angiocardiography, the additional irradiation must be considered. The image intensification technique is markedly advantageous in this situation also, as seen in the figures of Table 2. In addition it permits screening without full darkening.

TABLE 2. COMPARISON OF DOSES IN RADIOGRAPHY OF STOMACH

Six-valve apparatus—120 kv —1 mm Al filter—100 cm foc./screen distance—normal Bucky grid—thickness of object 21 cm

	Patient skin dose r
Normal radiography	0.5
Screen photography with a mirror camera	
(a) Single pictures, size 70x70 mm *	2 5
(b) Cinematography, 35 mm roll film,* 20 frames/sec during 1 minute	2000
Philips Image Intensifier and lens camera	
(a) Single pictures*	0.006
(b) Cinematography, 35 mm roll film,* 16 frames/sec during 1 minute	8

* Film Gevaert Ortho Scopix

Protection of the gonads by leaded rubber shields is recommended, although these should not be directly irradiated if correct primary masking of the exposure fields is done.

PICTURE QUALITY

The picture quality should enable the best possible diagnostic interpretation Until now, direct radiography has yielded the best results and the indirect methods have been inferior But since the optics of the photofluorographic devices have been improved they permit satisfactory resolution approaching that of direct radiographs The image intensifier also permits the use of fine-grain films, adding detail and sharpness to the picture. Primary masking of the exposure fields and suitable grids diminish scattered radiation Sharpness of outline demands immobility of the patient. Often narcosis and in certain cases controlled cessation of respiration are required

FILM PROCESSING

The roll films of direct radiography, photofluorography and image intensification techniques demand special darkroom devices for processing These rolls are developed with equal conditions for all the pictures The single films from the film changer can be developed with standard darkroom facilities under visual control. It is thus possible to correct within certain limits the devastating effect of under- and overexposures, as well as to get a desired variable contrast in different pictures from the same series, whereby both the well penetrated heart and the vascular lung pattern are visualized.

ECONOMY

The film changer, roll film device and photofluorographic apparatus of the Odelca type require approximately the same x-ray apparatus The investment costs are fairly equal for the corresponding recording devices As the image intensifier can operate at a lower milliampere output the investment costs for the x-ray apparatus are less for this unit.

The film costs are considerably less expensive for both indirect types than for the direct radiography in normal size. The film changer operates with standard single films, which may be cheaper and easier to obtain than the special roll film in large size.

POSITION OF THE RECORDING DEVICE

The position of the two recording devices for roll film radiography is in its present performance permanently fixed at a 90 degree angle. However, a free displacement of the picture fields under certain conditions is desirable and can be done with the three other types which are run as independent systems (Figs 7–9)

SYNCHRONOUS ELECTROCARDIOGRAM

The synchronous electrocardiogram with automatic exposure marking

TABLE 1 EXPOSURES REQUIRED FOR DIFFERENT TYPES OF RECORDING

A-P angiocardiogram of middle-sized adult at 1 m focal distance

Single exposure requires for:	kv.	80	100	125
Photofluorography, mirror image	mAs.	100	25	6
Direct radiography		30	8	2
Image intensification recording		0 8	0 3	0 1
(Exclusive exposure values, total radiation doubled because of uninterrupted x-ray tube discharges)				

exposure rate of 50/sec. Photofluorography with the Odelca device requires somewhat more milliamperage than direct radiography (Table 1).

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SYNCHRONOUS ELECTROCARDIOGRAM

The synchronous electrocardiogram with automatic exposure marking



FIG. 7. Free displacement of the picture fields is permitted with the film changer, where the two recording devices are run as independent systems

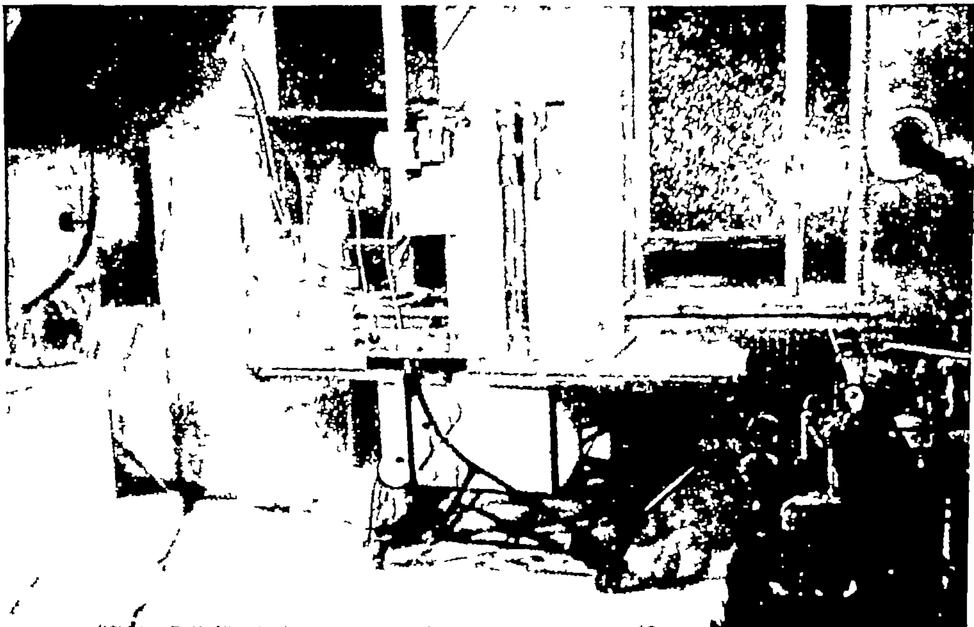


FIG 8. The film changer built into an operating table, to be used in connection with major operations (The University Clinic, Turku, Finland)

enables one to determine the time relationship of the single pictures reciprocally and to the heart cycle. This is necessary for the understanding of the cardiac dynamics (Fig 10).

CONTRAST MEDIUM

The desired properties are: (a) low toxicity; (b) good mixing ability with blood, correctly isodynamic, isotonic, isoviscous The amount of contrast



Fig 9 Same apparatus, adjusted for horizontal exposure.

medium must be adjusted to the heart size and be adequate for filling of one side of the heart. The speed of injection must be high enough to opacify only one side of the heart at a time.

INTRODUCTION OF THE CONTRAST MEDIUM

The optimal temperature of the contrast medium is 37° C. There are two modes of introduction, the peripheral or intravenous, and the central, intracardiac or into the great vessels through a catheter The choice must be made individually with respect to the information desired The intravenous injection is most useful in the study of the physiology and pathophysiology of the heart as a whole. The intravenous injection is made either in the upper or in the lower extremity, the choice being determined individually The intracardiac injection yields detailed anatomic visualization of limited regions By introducing the contrast medium directly into the heart, it is possible to get a good opacification of the cardiac chambers or vessels of interest in the

particular case. For diagnostic use it is often an advantage to restrict the examination in this way. For the anatomic study much can be gained by not having other heart cavities or vessels simultaneously filled which may project over and possibly mask the actual findings. It is of further advantage to get a high concentration of the contrast medium just at the strategic point. These requirements are best met with if the tip of the catheter lies in the chamber or vessel to be examined (Table 3).

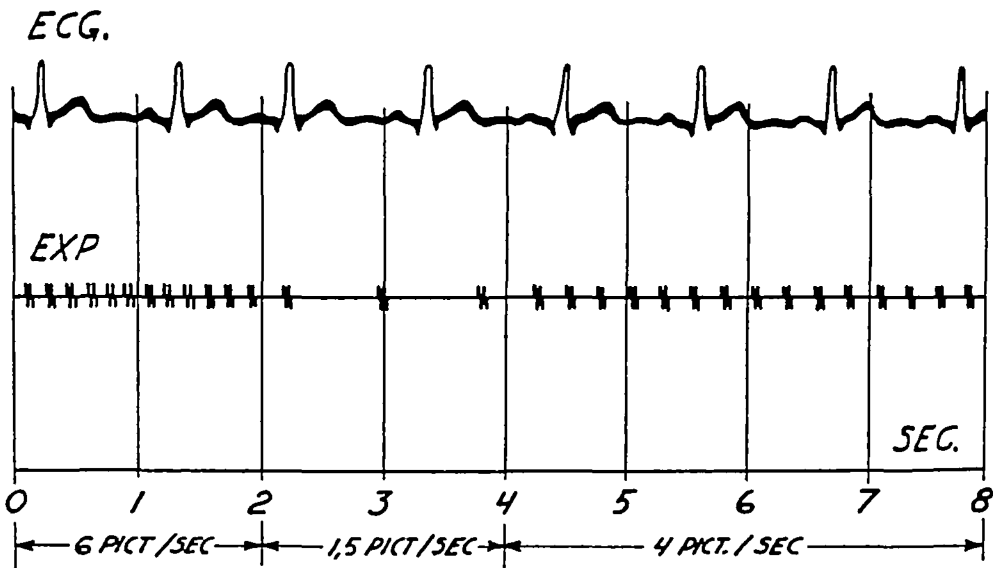


Fig 10 The synchronous electrocardiogram with automatic exposure marking. The exposure frequency, selectively varied, is recorded

TABLE 3 CONTRAST INJECTION IN RELATION TO EXPECTED DIAGNOSIS

Intravenous angiocardiography
The whole circulation through the heart and great vessels

Intracardiac (selective) angiocardiography

POSITION OF THE CATHETER TIP	EXPECTED DIAGNOSIS
Pulmonary artery	Coarctation of the aorta
" "	Anomalous pulmonary venous return
Left atrium or pulm artery	Atrial septal defect
Right ventricle	Ventricular septal defect
" "	Patent ductus arteriosus
" "	Isolated pulmonary stenosis
" "	Tetralogy of Fallot

Using this technique it seems to be possible sometimes to increase the pressure in the injected cavity and this way artificially create a shunt in cases of interventricular septal defects. By this method it is also possible to take full advantage of the results of the catheterization, considering the pressures obtained in the various chambers, the oxygen content of blood withdrawn from them and the roentgenologic aspects of the heart catheterization. Based on these results and the clinical examination, the injection of contrast can be

carried out in a way which gives optimal information for the diagnosis and an eventual operation.

Dr Jonsson, who has worked with the rapid biplane serial recording procedure, will demonstrate clinical examples achieved with this method

The injection through a catheter requires a special device for satisfactory pressure and speed The Gidlund injector, which also prewarms the contrast fluid, is an example of a far advanced automatic device (Fig 11)

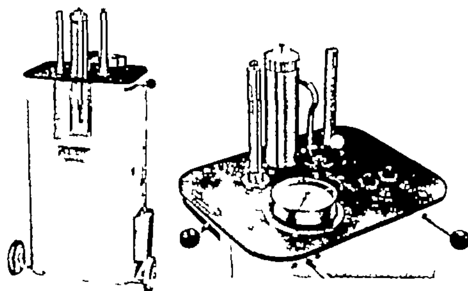


Fig 11 High pressure injection syringe for angiocardiography model of Dr Gidlund manufactured by Elema.

POSITIONING OF THE PATIENT

The optimal position of the patient has to be selected in the individual case on the basis of screening, anatomy shown in conventional radiograms, expected pathology, and so on. Fixed rules do not apply It is desirable to have in one projection the right and the left hearts quite independently projected It is also desired that there be no overlapping of the atria and the ventricles in the other projection. The x-rays, for these reasons, should follow in part the direction of the interventricular septum and in part that of the atrioventricular septum. This is best accomplished by utilizing the two oblique projections

PRESENTATION AND VIEWING OF THE PICTURES

Conventional x ray pictures are viewed and judged one by one. With biplane projection they are viewed in pairs and the entire series of films should be accessible for viewing at the same time. This is desirable in order to make comparisons of the onset and disappearance of the opacification in the different chambers of the heart, as well as variations in the density of the shunts This demands examination of the pictures of both sides of the heart at the same time. For this reason, sufficiently large viewing boxes are needed, on which all the pictures from an angiocardiogram may be mounted and ex-

amined. Separate, direct radiograms of normal size are favorable for this purpose. Films with diminished picture size must be copied and enlarged to permit detailed comparison of single pictures from different parts of the film strip.

To project serial pictures cinematographically, an exposure frequency of at least 16/sec. is necessary to give the human eye a perception of uninterrupted movements At present this frequency is not attainable by direct radiography or by fluorography. The technical difficulties of film transport have not been overcome in the former and the limits of safe irradiation will be exceeded in the latter, which demands a great primary x-ray output. In medical radiology therefore, the image intensification method offers the only means for roentgen-cinematography. At present, owing to the limitation of field size of approximately 10 cm. diameter, the method is restricted to objects of small size. In angiocardiography, it can be applied in the newborn and infants Our institute has demonstrated cinematographically the changes of the circulation at birth, the first breath, and other studies of the dynamics of the respiratory tract and the alimentary tract in the neonatal period.

By cinematographic projection of a film, the major cardiac movements, such as contraction modus, the changing declination of the heart axis and the pendulous motion of the atrioventricular septum, are impressively visualized. However, the events of cardiac dynamics often occur far too fast to permit a detailed perception by the human eye and brain For this reason also, comparative studies of significant single films from the film strip are desirable and require a sufficient detail and sharpness in the pictures.

DIAGNOSTIC VIEWPOINTS

Clinically, the most important contribution of the angiocardiographic examination has been the demonstration of the anatomy of congenital heart malformations. It contributes to general understanding of the problem and serves as a guide for the surgeon. The procedure is now used mostly in conjunction with catheterization, so that intracardiac instillation of contrast medium, resulting in an increased local opacification, will facilitate the demonstration of previously obscure anatomic features.

As will be shown, the morphologic appearance of the interior of the heart is altered considerably during the cardiac cycle. The synchronous electrocardiogram with exposure marking is therefore necessary to explain the dynamic changes from one picture to the next and to enable us to relate the findings to the heart work If this is realized in the diagnostic interpretation, the information gained by angiocardiography will be enriched by dynamic *physiologic* aspects *in addition* to the elucidation of anatomic abnormalities. The standards of size and shape, as well as the topographic outlines of the heart, represent in single pictures static abstractions from a dynamic process As a result of physiologic alterations the heart is subjected to continuous changes in its absolute and relative dimensions These dynamics are an expression of cardiac function

In consequence the heart, as visualized by angiocardiography, should be

judged as a living entity with both anatomic and physiologic aspects, mutually interrelated. Study of both normal and abnormal physiology is possible only so far as the recording device is adaptable. The borderline between normal and altered dynamics is subject to interpretation.

CONCLUSION

In our presentation we have to a great extent dealt with the technical problems of recording and general principles of diagnostic approach in angiocardiography This has been done intentionally, with the aim to explore all the possible areas for improvement of the procedure. As stated in the introduction, there is great need of additional information for the physiologist, the cardiologist and the surgeon In a procedure which involves so many problems, both in recording and in interpretation, the technical and professional details of performance are intimately related. Thus the desired end result—the most accurate diagnosis—will profit from progress in any of the many details considered here.

DISCUSSION

Bengt Jonsson (*Stockholm*)

For the preoperative diagnosis of congenital heart disease, both heart catheterization and angiocardiography are often required. Hemodynamics are best studied by catheterization, but angiocardiography will give precise information about anatomic details of importance to the surgeon. To get the malformation clearly outlined, the contrast medium should be injected as close as possible to the area involved. Heart catheterization is first performed, and on the basis of these findings it is decided where the contrast medium is to be injected. These examinations are, therefore, performed by a team of cardiologists and radiologists I have worked at Caroline Hospital in Stockholm together with Mannheimer, Kjellberg and Rudhe.

I will now present some examples from our series

In *pulmonic stenosis* the injection is made into the right ventricle. The tip of the catheter must be placed in the inflow tract or apex and not in the infundibulum in order not to overlook an infundibular stenosis With this method it is possible to demonstrate details in the outflow region of the right ventricle.

Figure 1 shows a valvular stenosis with the dome-shaped valves, the jet; the hypertrophy of infundibulum, which contracts at end of systole but dilates during diastole and gives no functional stenosis

Figure 2 shows an infundibular stenosis and the size of the third ventricle. In Fig. 3 is shown an associated ventricular septal defect, so often found in infundibular stenosis During diastole contrast medium is forced through the defect. Gas analysis may not detect this defect.

In *tetralogy of Fallot* the injection is also made into the right ventricle. The examination will give information about the anatomy of the infundibular

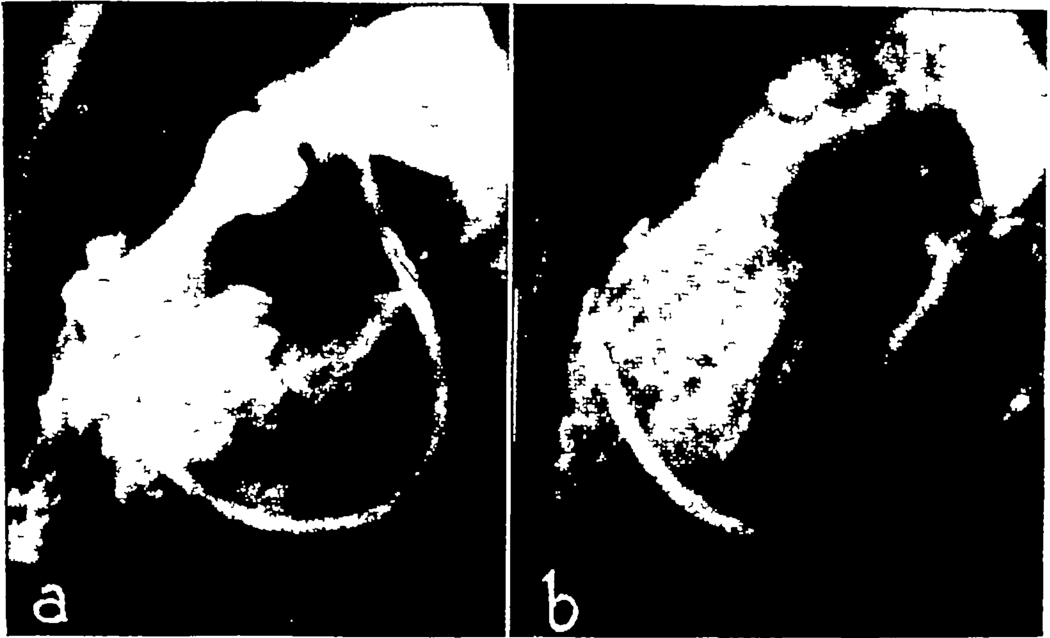


Fig 1 Visualization of valvular pulmonic stenosis. *a*, Systole, *b*, diastole.



Fig 2 Infundibular stenosis, with well developed "third ventricle"

stenosis and the size of the third ventricle, the size and position of the pulmonary artery branches, the degree of overriding of the aorta, the ventricular septal defect and the position of the subclavian. The frontal and lateral projections are preferable to oblique projections. The arteries are then best depicted. Also the defect and stenosis are best illustrated in this projection



Fig 3 Demonstration of interventricular septal defect in addition to infundibular stenosis

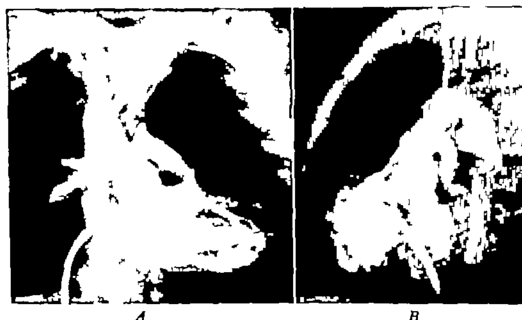


Fig. 4 Angiocardiography in the tetralogy of Fallot. *A* Frontal projection, *B*, lateral projection.

because the heart is clockwise rotated and the septum lying in the frontal plane (Fig 4)

A *ventricular septal defect* should be best visualized with injection into the left ventricle, but this is seldom possible. It was done only in one of our cases Direct visualization of the defect is also possible by means of injection

into the right ventricle if the systolic pressure is more than 50 mm. Hg in the right ventricle and if the injection is made very rapidly (less than 1 sec). The relation of the aortic root to the ventricular septum is best judged in the lateral projection (Fig. 5)

An *uncomplicated atrial septal defect* can be visualized only if the contrast

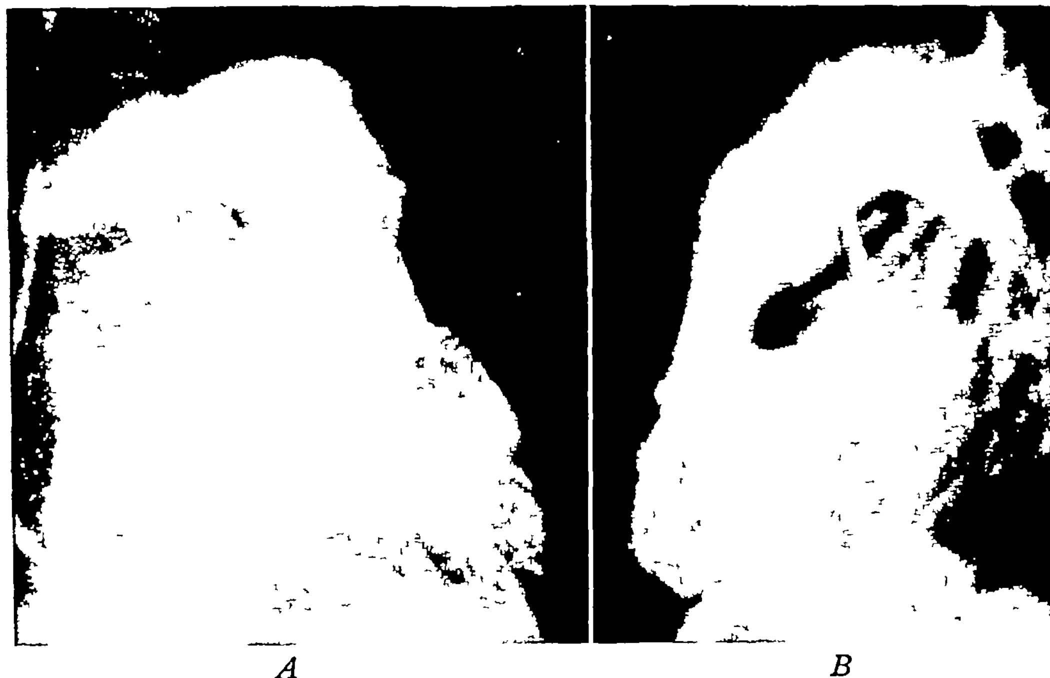


Fig 5 Demonstration of ventricular septal defect *A*, Frontal projection, *B*, lateral projection



Fig 6 Contrast medium injected into left atrium to show interatrial defect

medium is injected into the left atrium. Oblique projections are the best. It can be possible to differentiate between ostium primum and secundum and to exclude a mitral stenosis (Fig 6).

A *patent ductus arteriosus* is most clearly demonstrated by passing the catheter through it. If it is desirable to get information about anatomic details an aortogram may be done. Another method is to inject the contrast medium when the catheter is withdrawn from the aorta into the pulmonary artery (Fig 7)



Fig 7 Angiographic study of patent ductus arteriosus

Coarctation of the aorta is best demonstrated with aortography (Fig 8), but in children it is also possible to get good pictures if the injection is made into the pulmonary artery Sometimes it is necessary to give two injections, one into the aorta and another into the pulmonary artery, as in a case with complete aplasia of the aortic arch with the descending aorta supplied from a pulmonary artery through a patent ductus (Fig 9)

In *transposition of the great vessels* it is often necessary to inject both in the right and left side of the heart (Fig 10)

For the injection we use an automatic syringe which allows a rapid injection. It is so constructed that air embolism is entirely eliminated. We prefer a catheter with thin wall and wide lumen, the Lehman catheter To avoid recoil of the tip of the catheter by the pressure of injection we have bored holes in retrograde direction in the wall a bit from the tip

Before the contrast medium is injected into the right ventricle it is important to be absolutely certain of the position of the catheter to avoid injection into a coronary vein. The catheter is first advanced into the pulmonary artery or the aorta and then withdrawn into the ventricle under fluoroscopic



Fig 8 Angiographic study of coarctation of aorta The collateral vessels are well shown



Fig 9 Demonstration of aplasia of aortic arch, with descending aorta being supplied by patent ductus arteriosus *A*, Injection of proximal aorta, note sinuses of Valsalva and coronary artery *B*, Injection of pulmonary artery by catheter A part of the pulmonary artery continues smoothly as the descending aorta.



Fig 10 Demonstration of transposition of the great vessels. *A* Injection of right ventricle, with filling of aorta and branches *B*, Injection of left ventricle with filling of pulmonary arteries

control A small quantity is then injected under fluoroscopic control as a test dose.

During the examination, general anesthesia is used and succinylcholine is given to produce complete muscular paralysis Artificial respiration is given with pure oxygen and the angiocardiography is performed during apnea in inspiration.

Tyge Sondergaard (*Aarhus, Denmark*)

I want to discuss only one side of this problem, diagnosis of the atrial septal defect. I want to stress immediately, that I am not sure this method is safe yet to use clinically It is only experimental so far, but we have operated upon 78 dogs, two of them dying from causes unrelated to the procedure, and we are still carrying on

First we produce atrial septal defects under direct vision with the use of the double ring clamp and place brain clips of the Cushing type in the edge of the defect in order to be able to see where we have the defect on plain x-ray pictures In Fig 1 we have injected the contrast medium into the right atrium. The clips are visible and the defect is outlined.

In Fig. 2 we again see the defect beautifully outlined, with filling of the left atrium, and retrograde filling out in the pulmonary veins You can also see filling of the hepatic veins very clearly

We anesthetize the dog and through an intratracheal tube we apply 40 to 50 mm. pressure on the lungs By this method we block the pulmonary artery and the pulmonary circulation completely for 10 seconds, which keeps out the contrast medium from the pulmonary artery The only way the contrast medium can escape is through the defect.



Fig 1.



Fig 2

Fig 1. Atrial septal defect in dog Contrast medium injected into right atrium SCV superior caval vein ICV inferior caval vein LA left atrium

Fig 2 Filling of left atrium, retrograde filling of pulmonary veins, and filling of hepatic veins.



Fig 3

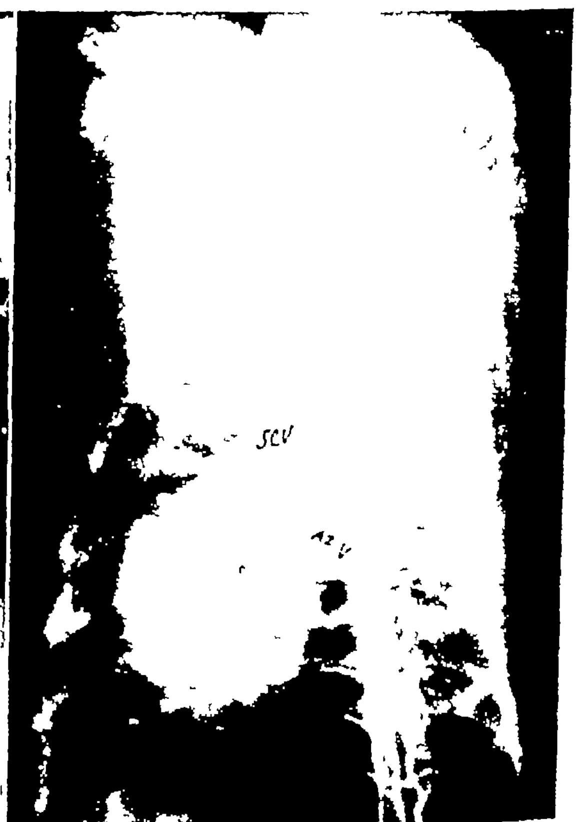


Fig 4

Fig. 3 Edge of defect not clearly visible Clips are covered by contrast medium

Fig 4. Contrast medium injected just opposite azygos vein SCV. superior caval vein. Az.V azygos vein

In Fig. 3 the defect cannot be seen clearly and the clips are covered by the contrast medium. Again you can see filling retrograde of the veins

Figure 4 is an x-ray photograph in which we have injected the contrast medium just opposite the azygos vein. As there are no valves in the azygos vein system along the spine, you can see there is complete filling of all the veins in the spinal canal. The brain surgeon is interested in that part of it.



Question Does pulmonary hypertension increase the risk of selective angiocardiography?
Answer (*Dr Lind*) My answer to this question is "No"

Question Does selective angiography, injection of dye into the vessels, increase the risk of angiocardiography?

In September, Swedish rumor had it that the addition of procaine or related substances to the injected contrast medium would diminish the sensitivity of the myocardium if the contrast medium should enter the coronary vessel Is this rumor well founded? If so, have the original hopes been borne out?
Answer (*Dr Lind*) At Karolinska we have not used this method. We have not found that the selective procedure increases the risk of angiocardiography as compared with the intravenous Therefore, it is difficult to judge the value of procaine.

Question What contrast medium is advised?
Answer (*Dr Jonsson*) Concerning the contrast medium used, we now use Urokon and it seems to cause less reaction. We always give test doses before the injection of the contrast medium. I remember one patient who has been in our hospital three or four times We have not been able to perform angiocardiography on this patient because he shows signs of reaction.

Question How much dye is injected directly into the heart, in the work which has been presented here?
Answer (*Dr Jonsson*) Concerning the dosage, we do not like to give more than 1.2 cc. per kilogram of body weight. It depends upon the size of the heart and the size of the shunt, but we do not like to give more than this dose under any circumstances

Question If you have selected the point, how often do you have to repeat the angiocardiography, injecting at a different point?
Answer (*Dr Jonsson*) Regarding repeated examinations, I think there is absolute contraindication to repeated examinations on the same day We had one such case, and the patient died. We have to wait at least a week between injections Of course, if you want to have a detailed

anatomic study of a very severe and very complicated malformation, you have to do several injections both in the right ventricle and in the left ventricle, and so on. In most cases it is not necessary, but if you work with babies there are some very complicated cases in which there is no alternative.

Question: I would like to ask the Swedish group if they have had any experience with direct percutaneous injection of contrast media into the ventricular chambers.

Answer (Dr. Jonsson): I have had no experience with direct injection of the heart. Bjork, at Sabbatsbergs Hospital, has injected with puncture from the back directly into the left atrium, especially in cases of mitral valvular disease. He puts in a polyethylene tube so that he can catheterize the ventricle and atrium He can inject in the left ventricle to see if there is also a lesion there.

I should like to mention a new technique performed by Hansson for heart catheterization, with a balloon catheter, whereby he can close one or the other side of the pulmonary artery and study the results from the point of view of cardiac hemodynamics.

Chairman Holman: Dr. Jonsson, did you say that you injected the dye slowly or rapidly?

Dr. Jonsson: Rapidly.

Question (Dr. Richard J Bing). I would like to ask the Swedish workers a question, if I may. In some of those studies which we have done, we found that intra-aortic catheterization with injection of Thorotrast was quite a dangerous procedure. We also felt that in some work on the dogs we have done, regional angiocardiography, the injection of Thorotrast through a catheter into the right ventricle might be hazardous.

I would like to ask them what their risk was in their procedures, because the risk apparently comes not only from anoxia but also from the fact that the dye or the iodine may reach the central nervous system in very large concentrations, and may produce irreparable damage and coma, with the patient dying in several days.

I do not wish to detract from the beautiful work they have done, but we are simply afraid of the intra-aortic catheterization with the injection of iodine, and we would like to know what their results have been in this respect.

Answer (Dr. Jonsson) First, about thoracic arteriography, the technique is always very important. It is important to know exactly where the tip of the catheter is placed. It must not be so low that the contrast medium goes down to the coronary arteries. It is a big risk to have it so high up in the ascending aorta that the tip of the catheter will be thrown up into the anterior coronary artery. We hold it so that the tip is in the right grade. There is less risk of the catheter being pressed backward and slipping up into the coronary arteries.

I work mostly with children, and with them we do not have so many cases of thoracic arteriography It is possible to get good pictures with injection into the pulmonary artery In Stockholm we have performed 300 or 400 thoracic arteriographies There was one death. The patient was an old man who died before the injection, during anesthesia In another case there was cerebral damage. We went back after a week, without any trouble. Too much contrast media had gone up into the head and brain because the catheter had slipped up The fault lay in the poor position of the catheter

It is very important to have the patient under good anesthesia and very well saturated with oxygen before the injection. My own experience with children is very good. We have had no deaths

Concerning the other question about angiocardiographies, if we discuss risks we must remember that they vary greatly depending upon how severely ill the patient is We have had patients come to our laboratory in such bad condition that we thought they would die while we were taking the electrocardiogram If they had died then, nobody would say it was the fault of electrocardiography

In some patients we have estimated that we would do the angiocardiography on a Tuesday, but for some reason it was not performed, and the patient died at the hour of the day when we had intended to do the angiocardiography If we *had* performed it on Tuesday, of course, it would not have been the actual cause of the death. In one such case we were happy that we had not performed it, because the patient died at the time we had planned to do it.

Sometimes we are very surprised at how bad the patient is before examination, and how good he looks afterwards I must say that we have had a few deaths The first one was a case with overriding, and at that time we did not have the new injector It was not possible to be quite sure about air embolism with this metal syringe After that accident we changed our apparatus and have had no trouble since.

We have had two other deaths, but they were severely ill patients with heart decompensation. In one case of severe heart decompensation, the examination was performed just because the parents wanted to have everything possible done for the child.

PULMONIC STENOSIS

WILLIS J POTTS (*Chicago*)—MODERATOR

PANEL ON THE DIAGNOSIS AND TREATMENT OF PULMONIC STENOSIS

WILLIS J. POTTS (*Chicago*), MODERATOR

SIR RUSSELL BROCK (*London*)
CHARLES DUBOST (*Paris*)
GUNNAR EKSTROM (*Stockholm*)
JOHN KEITH (*Toronto*)
RODOLFO KREUTZER (*Buenos Aires*)
HENRY SWAN (*Denver*)
HELEN TAUSSIG (*Baltimore*)

DR. POTTS

Pulmonic stenosis is perhaps the most important congenital cardiac malformation, and occurs in a variety of forms. The morbid anatomy is subject to great variation In some instances the stenosis is valvular, in others it is infundibular, and still in others both the infundibulum and the valve are involved

This variation in the pathology in the region of the valve, together with the accompanying lesions elsewhere in the heart, produces a number of distinct clinical entities Much progress has been made in both diagnosis and surgical management of these conditions, yet diagnosis is often difficult, and even when it is clear, there is a lack of agreement as to the surgical treatment of choice Hence, it seems that congenital pulmonic stenosis is a subject worthy of discussion

Of the eight members on the panel, three are pediatric cardiologists and five are surgeons. Inasmuch as diagnosis must precede surgery, I will call first on the diagnosticians. Dr Helen Taussig will be the first speaker.

DR TAUSSIG

Dr. Potts has asked me to review with you briefly the differential diagnosis between tetralogy of Fallot and pulmonic stenosis with an intact ventricular septum

In tetralogy of Fallot, at birth the infant may or may not be cyanotic The cyanosis usually increases It commonly does not occur until 4 to 6 months of age, and may not appear until the infant starts to walk.

In pulmonic stenosis, if there is cyanosis at birth, usually that cyanosis disappears and does not recur until 2 to 5 years of age When cyanosis does occur in pulmonic stenosis with an intact septum, it occurs insidiously and is not associated with paroxysmal dyspnea or loss of consciousness, whereas an

infant with tetralogy of Fallot usually suffers from episodes of cyanosis and from attacks of paroxysmal dyspnea which may or may not progress to loss of consciousness During infancy he may have many such attacks He prefers to lie in a knee-chest position. When he is older and learns to walk, he squats when he gets tired In contrast to this, in pulmonic stenosis with an intact septum the child does not lie in the knee-chest position, and does not squat when tired.

In tetralogy of Fallot the infant is usually poorly developed and slow in weight gain. The child with pulmonic stenosis and an intact septum is a strong, sturdy infant who gains well.

The difficulty with pulmonic stenosis is the increased work in the right ventricle, which may lead to progressive cardiac enlargement, engorgement of the liver, pulsation in the liver and cardiac failure. The infant with tetralogy of Fallot suffers from hypoxia. The overriding aorta acts as an escape mechanism, so the pressure of the right ventricle is not as high. The condition does not lead to progressive cardiac enlargement. His danger is from hypoxia and polycythemia, and he is liable to die from oxygen lack and not from cardiac failure.

During physical examination, one will find also that in tetralogy of Fallot the murmur is inversely proportional to the severity of the stenosis Usually there is a harsh systolic murmur along the left sternal border and an audible second sound, whereas in valvular pulmonic stenosis there is a harsh systolic murmur and a weak or absent second sound. As the heart enlarges the murmur is heard higher and higher up over the second left interspace, and then it abruptly ends and one hears nothing at all, the second sound is absent.

Again, under the fluoroscope, in tetralogy of Fallot the heart is normal in size, the lung fields are clear There may be small pulmonary arteries visible There is a concave curve at the base of the heart. In valvular pulmonic stenosis, the heart may be normal in size or it may be enlarged There is fullness of the pulmonary conus The pulmonary arteries are prominent, but the periphery of the lung fields remains clear

In tetralogy of Fallot the electrocardiogram usually shows right axis deviation and right ventricular hypertrophy, but it does not show the extreme right axis deviation or the extreme evidence of right ventricular hypertrophy—what is commonly spoken of as right ventricular strain path in the precordial leads Therefore, I think that in most instances a simple clinical history, physical examination, x-ray, fluoroscopy and the electrocardiogram will suffice to differentiate a patient with tetralogy of Fallot from one with valvular pulmonic stenosis with an intact ventricular septum.

DR. POTTS

Dr Rodolfo Kreutzer will present his classification of the types of pulmonic stenosis, excluding the tetralogy of Fallot.

DR. KREUTZER

From an anatomico-clinical point of view, the following four groups are distinguished.

GROUP I: VALVULAR PULMONIC STENOSIS, "DOME-SHAPED TYPE," WITH CLOSED INTERVENTRICULAR SEPTUM This group is far more frequent than the other three together, and in it we can recognize four types:

Type A: Mild valvular pulmonic stenosis with or without interatrial septal defect. There is no dyspnea or cyanosis. A loud systolic murmur with a palpable thrill is always present, occasionally accompanied by a mild diastolic murmur. The pressure gradient between the right ventricle and the pulmonary artery is small. The electrocardiogram can be normal. We do not believe that valvotomy is necessary and we consider it contraindicated when the shunt from left to right (in case there is an interatrial communication) is marked.

Type B: Severe pulmonic stenosis with both septums closed. There is progressive cardiac enlargement and early congestive cardiac failure. There is no cyanosis The pressure in the right ventricle is very high and surpasses the systemic pressure. Valvotomy is clearly indicated

Type C: Severe valvular stenosis with persistence of the foramen ovale. The symptomatology is similar to that of the tetralogy of Fallot. As the child grows the pulmonary arch becomes more prominent, the heart enlarges and the electrocardiogram shows right ventricular strain. There is increasing dyspnea and cyanosis. Here also, the valvotomy is clearly indicated.

Type D: Severe valvular stenosis with large interatrial septal defect. The radiological picture is identical with that of the atrial septal defect but the symptomatology is similar to that of the tetralogy of Fallot. As in Type C, the angiocardiography may show early filling of the aorta from passage of the contrast medium through the atrial defect. The concentric hypertrophy of the right ventricle and the reduction of the size of its chamber could theoretically be a handicap for a successful valvotomy.

GROUP II: VALVULAR PULMONIC STENOSIS, "DOME-SHAPED TYPE," WITH INTERVENTRICULAR SEPTAL DEFECT. This group is usually mistaken for Roger's disease Angiocardiography does not show the simultaneous opacification of the aorta and the pulmonary artery. The catheter can enter the aorta through the ventricular septal defect The pressure in the right ventricle is the same as in the aorta Valvotomy would be indicated if the left to right shunt is small.

GROUP III: SCARRING VALVULAR PULMONIC STENOSIS WITH CLOSED INTERVENTRICULAR SEPTUM. The cusps are retracted against the arterial wall, and there is also a reduction of the size of the valvular ring The pressure in the right ventricle is very high Valvotomy cannot be accomplished in this situation

GROUP IV: STENOSIS OF THE MAIN TRUNK AND/OR OF THE PRINCIPAL BRANCHES OF THE PULMONARY ARTERY WITH CLOSED INTERVENTRICULAR SEPTUM. The anatomic lesion is located in the trunk and in the main branches of the pulmonary artery and consists of a panarteritis which almost occludes the lumen of the artery. Surgical treatment is not feasible.

DR. POTTS

Dr. John Keith will present some of his experiences with angiocardiography in the study of pulmonic stenosis

DR. KEITH

Angiocardiography has a special niche in the study of pulmonic stenosis since it may give visual evidence of the site of obstruction It is useful as a separate method of investigation in these cases, but it is also helpful when used as an adjunct to cardiac catheterization, especially in selective angiocardiography when the injection is made with the catheter in a particular chamber of the heart.

In pulmonic stenosis, we are interested in the presence or absence of

1 Pulmonary valve or artery atresia
2 Valvular stenosis
3 Infundibular stenosis
4 Combined infundibular and valvular stenosis

A summary of the post-mortem findings of tetralogy of Fallot in the literature shows the following frequency of these types

Pulmonary atresia	20%
Valvular stenosis	10%
Infundibular stenosis	48%
Combined valvular and infundibular stenosis	22%

Pulmonic stenosis with or without a shunt through the foramen ovale provides a further grouping, and infundibular stenosis as an isolated defect can also occur but is rare.

Pulmonary atresia is not an uncommon finding since it occurs in 20 per cent of the post-mortems done on tetralogy of Fallot, and it is important to make the diagnosis since operations of the direct type will not be successful or at least will be much more difficult when this form of obstruction occurs Furthermore, the shunt operations are not feasible unless there is an adequate pulmonary artery to accept the flow from a systemic artery As direct operations become more frequent, a detailed knowledge of the pathology of the outflow tract of the right ventricle, the pulmonary artery, becomes more and more imperative. *Angiocardiography perhaps plays its most useful* purpose in visualizing this type of obstruction since it may be difficult to clarify its presence by the cardiac catheter

Figure 1 demonstrates pulmonary atresia in a 3-month-old baby with a large right ventricle and the aorta filling from it. The atresia is obviously valvular and the angiogram shows quite clearly the blind end of the infundibulum of the right ventricle. Figure 2 shows another instance of tetralogy of Fallot with an overriding aorta, the aorta filling from the right ventricle, and a small blind tract in the muscle of the right ventricle at the site of the infundibulum. In pulmonary atresia it is useful to have an angiocardiogram from a lateral view, since it clearly shows the lack of filling of any pulmonary artery from the right ventricle and at the same time reveals the large collaterals coming off the aorta. It also delineates the size and position of the pulmonary artery that is present and aids the surgeon in making up his mind as to whether a shunt operation is feasible.

Another type of atresia that is well visualized by angiocardiography is that in which there is atresia of the pulmonary artery to one lung, as is shown in Fig 3 Here there is obstruction to the flow in the pulmonary artery as it

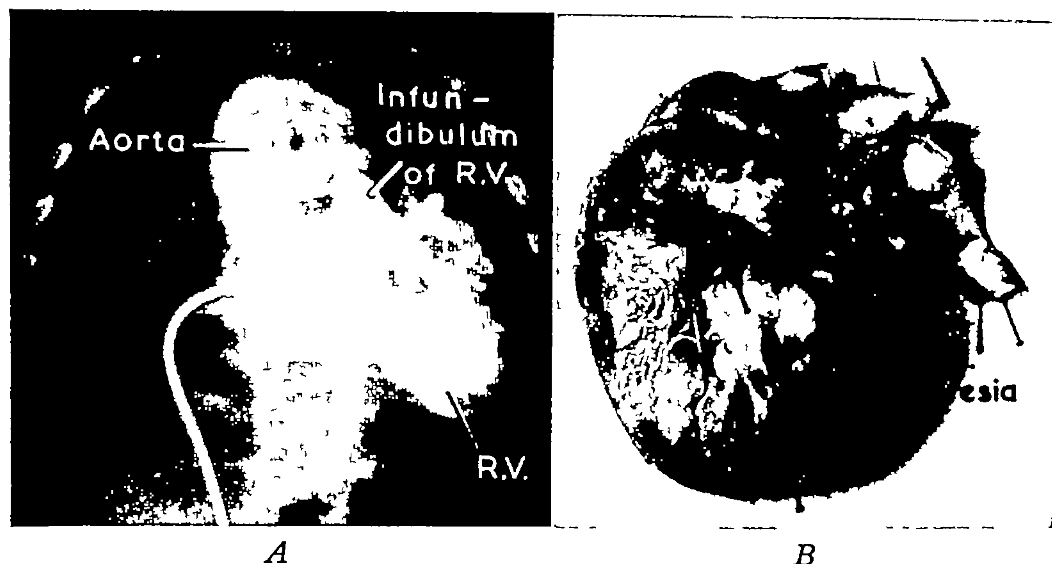


Fig 1 Pulmonary atresia in an infant of 3 months *A*, Angiocardiogram *B*, Post-mortem specimen



Fig 2 Angiocardiogram showing right ventricle ending in a blind tract

advances into the right pulmonary artery, and there is absence of the left pulmonary artery This type of defect may be suggested by the plain x-ray and by the position of a murmur to the right of the sternum, but it is best delineated by angiocardiography.

Figure 4 shows the pathologic anatomy of *pulmonic valvular stenosis*, the cone-shaped valve with the small opening in the center This 7-month-old baby died before operation could be performed This cone type of valvular



Fig 3 Angiocardiogram showing atresia of left pulmonary artery



Fig 4 Post mortem specimen of typical dome-shaped deformity in valvular pulmonic stenosis To illustrate the technical problem in the operation for correction of the condition, the smaller valvulotome was passed through without producing a cut and the larger made a cut only $\frac{1}{8}$ inch in depth on one side

stenosis can be shown clearly by angiocardiography, especially in the lateral view (Fig 5)

When valvular stenosis with a patent foramen ovale is present and angiocardiography is performed by an injection into the venous system or right atrium, contrast medium usually passes through from right atrium to left atrium, and from the left ventricle out into the systemic circulation. There

may be very little contrast medium seen in the pulmonary tree, if the stenosis is marked. However, a selected angiogram with the catheter placed in the right ventricle almost invariably reveals the pulmonic stenosis.



A B

Fig 5 Angiocardiography in pulmonic valvular stenosis *A*, Systole, note distention of the dome-shaped valve *B*, Diastole, note that the pulmonary valve is pressed toward the ventricle



A B

Fig 6 Angiocardiography in tetralogy of Fallot *A*, Anteroposterior view demonstrating infundibular stenosis *B*, Lateral view, showing valvular as well as infundibular stenosis

In *tetralogy of Fallot* one usually needs to have information about the infundibulum of the right ventricle as well as the pulmonary valve An example of angiocardiography in tetralogy of Fallot is shown in Fig. 6. This technique is only possible when the selective angiocardiogram is done and the contrast medium injected into the right ventricle The overlapping shadows of the right atrium do not interfere with a clear visualization of the defects

present. It should be pointed out, however, that the advantage of doing a selective angiocardiogram is that pressures can be obtained in the pulmonary artery, infundibular area and right ventricle so that the degree of obstruction can be determined on the basis of pressures This is usually more accurate than the information given by the angiocardiogram since a great variation in the size of the infundibular channel may appear in different phases of systole or diastole in the angiogram. The same is true of the valve opening Although one can make a diagnosis of obstruction at either of these sites, the cardiac catheter gives a more accurate appraisal of the degree of obstruction. The angiocardiogram on the other hand outlines the position of the infundibular stenosis, the size of the infundibular chamber and the size of the pulmonary artery in a manner that is not possible by other techniques

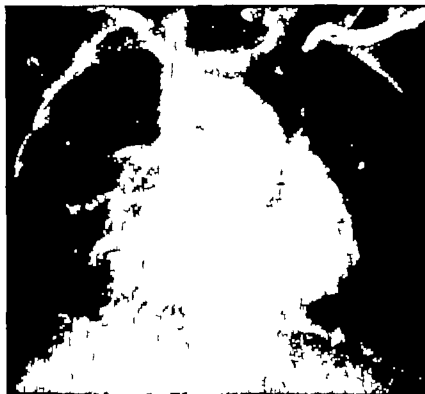


Fig 7 Angiocardiogram showing dextrocardia and pulmonic valvular stenosis

Another type of case that is well studied by angiocardiography is *dextrocardia*. Here the cardiac catheter has considerable limitations in that it is difficult to know exactly what chamber one is in, and in fact it is difficult to guide the catheter around from one chamber to another In Fig 7 is shown a film of a baby of 2 years with dextrocardia, there is no evidence of infundibular obstruction shown in the x-ray, but a small jet through a valvular stenosis appears to be entering a dilated pulmonary artery This child was considered to have valvular stenosis and was operated on successfully

Isolated *infundibular stenosis* may be recognized either by cardiac catheterization or by angiocardiography (Fig 8) If the narrowing is close to the pulmonary valve it may be difficult to tell by cardiac catheterization but the angiocardiogram may show clearly the presence of an infundibular stenosis, and that the valve is not involved.

It has been our experience that the angiocardiogram has been most useful in clarifying the problem of pulmonic stenosis, especially where pulmonary atresia is present, or dextrocardia, or a high infundibular stenosis with tetralogy of Fallot. The procedure in our experience is best done through a cardiac catheter so that one combines the information obtained by cardiac catheterization with that obtained by the angiocardiogram.

Perhaps the chief value of angiocardiography in pulmonic stenosis lies in the first few weeks or months before the usual diagnostic features of these congenital defects have had time to develop.

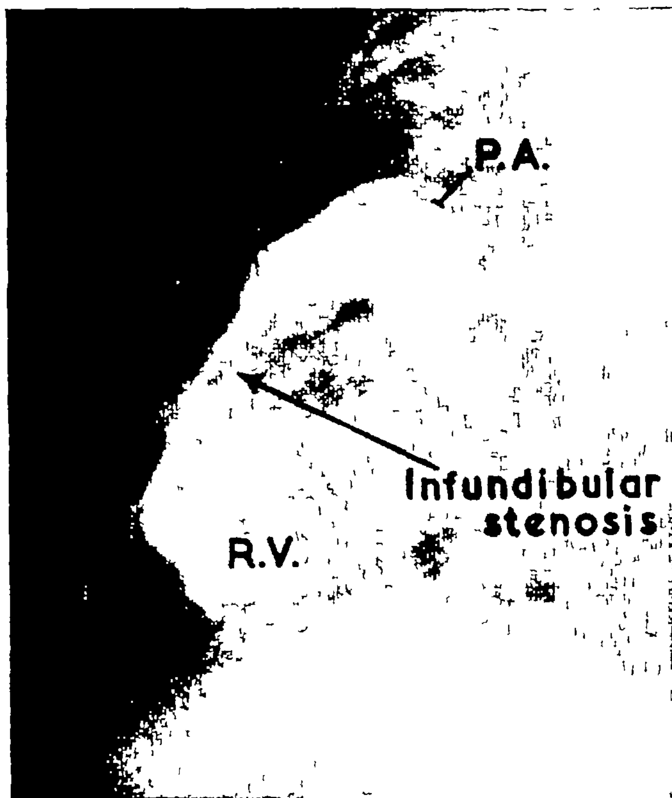


Fig 8 Angiocardiogram showing isolated infundibular stenosis

As the direct operations for relief of pulmonic stenosis come increasingly to the fore, as appears likely in view of the work of Brock, Swan and Lillehei, the need for early and accurate diagnosis will become increasingly imperative Angiocardiography in the early weeks of life will be found to fill a useful function in this regard.

DR. POTTS

I have been asked to speak on the shunt operation and interim results.

Sufficient time has passed since the field of surgery for cyanotic heart disease was opened by Blalock and Taussig to give a reasonable accounting of what can be accomplished by the shunt operation, what the hospital mortality is, and what the interim res are.

From our experience with 00 operations for cyanotic heart disease, we ha to a few c which are subject to change without

notice. I shall speak first about hospital mortality—how many patients survive, and thus have an opportunity of being more than a later statistic.

Our figures about to be presented are those from the very first case operated on in 1946 There is a 15 per cent mortality in children aged from 2 weeks to 3 years of age If the child can survive to 3 years, then the mortality promptly drops, and it has varied from 2.8 to 4 6 per cent. It has been our policy to refuse operation to no child, regardless of age or condition, if it can be demonstrated that there is a diminished flow of blood to the lungs

When we come to the pure isolated pulmonic stenosis case, the mortality has dropped. Again we have had a rather large number in the small age group from 23 days to 3 years, and from 3 to 16 years there has been no mortality, or a mortality of 1 6 per cent in the entire group

Now I want to say something about the follow-up results on the first 100 patients We were very fortunate in being able to follow every one who survived operation, although they came from many states and foreign countries. Thirty-nine come from the age group from one month to and including 36 months We have included 68 patients in group 1, those with good results They go to school, they run and play and get into fights, ride their bicycles, and so on.

Group 2 are the fair There were 16 of them. These are the children who do not do as well I might say that in the first group of 68 every patient had a continuous murmur In the second group of 16, all had a continuous murmur but one. They are somewhat cyanotic; they have to rest more frequently, and some of them go to special schools, and that sort of thing

The child with the poor result is a youngster operated on at age 13, who did very well for a while. Her heart became very large. She has been in and out of failure. That operation was done a little over eight years ago

Group 4 shows the unchanged. That child had an aortic-pulmonary anastomosis and also a subclavian pulmonary anastomosis Both have closed.

Five died after leaving the hospital, two from pneumonia, one of brain abscess, one of heart failure, and one of rheumatic fever The hospital mortality was 9 That is the total of the first 100 patients operated on between six and eight years ago

In conclusion, as a result of our analyses of mortality in general and interim results for six to eight years, we believe today that a patient with tetralogy of Fallot should have an aortic-pulmonary anastomosis on the left side if the arch curves to the left, and a subclavian pulmonary anastomosis also on the left side if the arch curves to the right.

Infants below one year of age, who must be operated on, should have an aortic-pulmonary anastomosis, regardless of which way the arch of the aorta curves

SIR RUSSELL BROCK

Obviously my task is made even more difficult by the masterly presentation which we have just heard by Dr Potts He has exemplified the good operative results and the good clinical results of the indirect procedures, and in the very short time at my disposal I have to contrast the results of direct operation.

I think the first thing to do is to look at this problem from an historical background. The initiation of the indirect operations for the relief of pulmonic stenosis dates from 1945, at the time when no operations were being done on the structure of the heart itself—or none of any importance. The success of these operations was immediate and, of course, of historical importance.

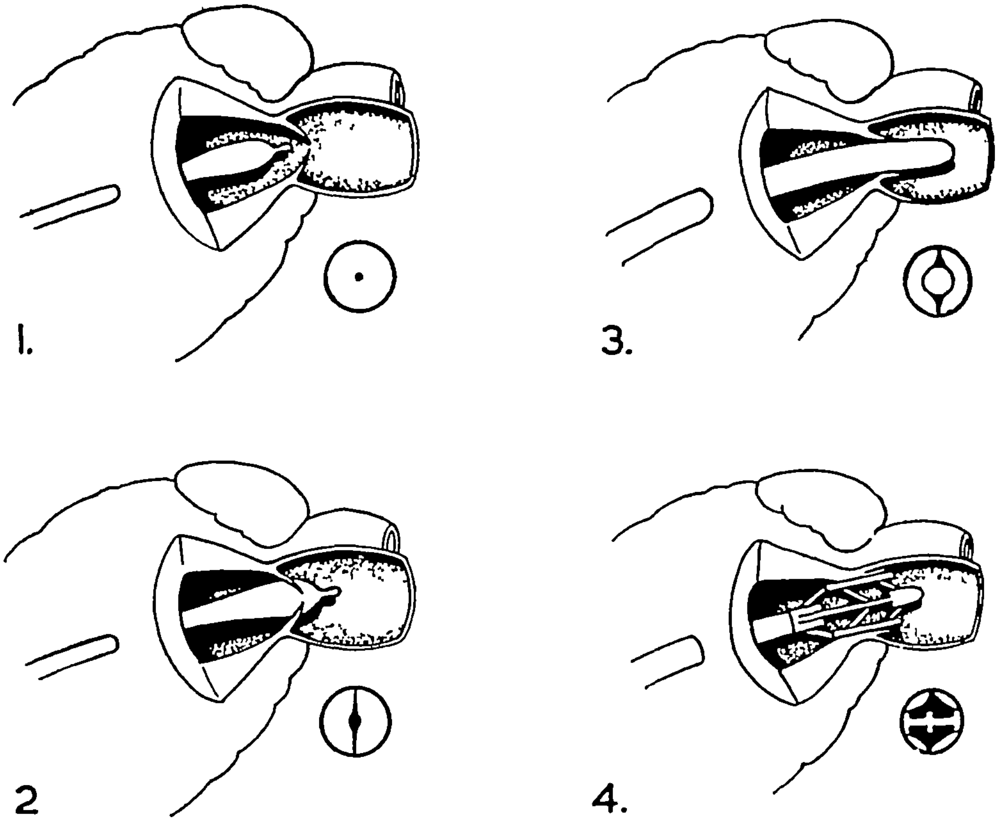


Fig 1 Transventricular pulmonary valvotomy

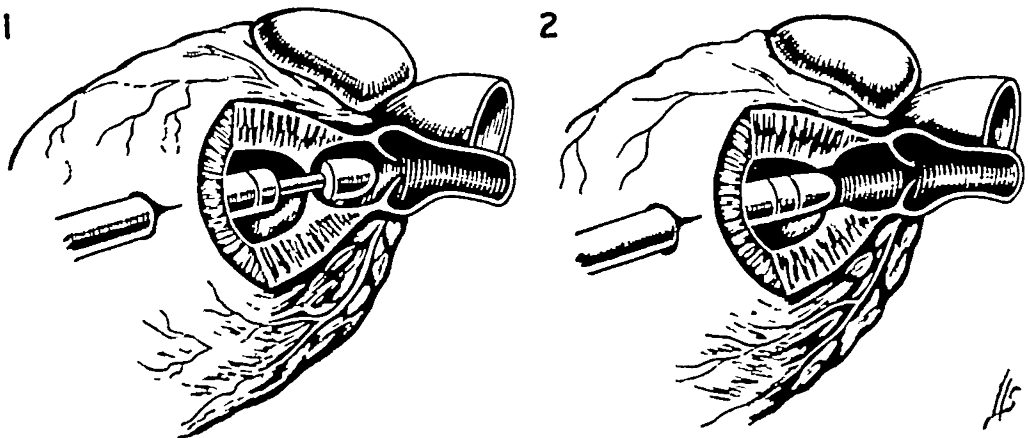


Fig 2 Punch infundibular resection

From 1945 to 1955 we have witnessed a complete change in the development of the surgery of the heart Today the development is entirely toward operating on the lesion itself, and I think we must take this into account in comparing these two forms of treatment. The future of all cardiac surgery must turn more and more toward direct and away from indirect surgery

There are various objections to the indirect procedure, but the chief one is that it is indirect, that it is a symptomatic method of treatment and does nothing to correct the heart abnormality

After the anastomosis has been going for a number of years, there is strong evidence to show that the obstruction, far from being relieved, continues to increase, and the effects of the strain on the right ventricle also worsen. Ultimately, if he survives long enough, we have a patient in whom there is virtually anatomic or physiologic pulmonary atresia, and he is living on the anastomotic circulation That is a condition which I do not think is compatible with a long, trouble-free life

There is no question of the brilliant success of the anastomotic procedure in a tiny child who is blue and who is doomed to die soon unless something is done to relieve it, particularly if the condition is an atresia, say a tricuspid or pulmonary atresia. What worries me is the older child who has reached a certain stage of physical and mental development and who in the future, if given a chance, may occupy a useful and happy position in the world

I find it difficult, in discussing this problem with parents and with patients, to be entirely happy about the future if an indirect operation is performed, however successful it may be. I cannot believe the ultimate prognosis is good.

The objections made to the direct operations at present are that they are difficult, that they are dangerous, and that the results are poor I have yet to see any satisfactory and comparable series of figures published by people who make these statements My own series consists of 140 cases of direct operation for tetralogy of Fallot (Figs 1 and 2). I want to say just a word or two about them

TABLE 1 FALLOT'S TETRALOGY COMPARATIVE RESULTS IN 140 CASES

	Very good and good	*Improved*	*Not improved*	*Died*
	/	/	/	/
Valvotomy 50 cases	74	12	2	12
Inf resection 62 cases	80	7	—	13
Combined inf resection and valvotomy 28 cases	72	21	—	7

In Table 1 you can see the relative numbers in these 140 cases This gives the comparative results and comparative mortality You can see, incidentally, that the results are good in valvotomy In all three cases the results are good

Table 2 shows the result in 140 direct operations done by myself, 165 indirect operations chiefly done by myself, and 857 indirect operations reported by Dr Taussig and Dr Blalock.

All I want to show from this table is that the mortality in the direct group, 11.5 per cent, compares very favorably with the mortality in my own series of indirect operations, 8.5 per cent, and in those of Dr Taussig and Dr

Blalock, 14 per cent. I have already shown you that clinical results compare well In other words, I think this experience in my own operative series shows that the mortality is not excessive and the results are satisfactory.

I don't think there is really any argument about the merits of these two procedures. I believe it has gone beyond the phase of arguing which should be done. There is no question that a good direct operation can give a perfect result, and the prognosis may be excellent.

The drawback that I find in doing the direct operation is that you may do too much and give the patient a slightly increased pulmonary blood flow or a considerably increased pulmonary blood flow. Clinically they are excellent, but one is unhappy because of the increased flow, one is unhappy because the heart may increase in size, and because signs of right ventricular strain may occur on the electrocardiogram, as indeed they also occur in a proportion of cases after an anastomotic procedure.

TABLE 2 FALLOT'S TETRALOGY

Results in { 140 Direct Operations; 165 Indirect Operations; 857 Indirect Operations }

	Direct		Indirect	
Result	*Number*	*%*	*Campbell, 165*	*Taussig, [illegible]*
Very good or good	107	76	77 5%	80%
Improved	16	10 5	8 5%	3%
Not improved	1	0 7	5 5%	2%
Died	16	11 5	8 5%	14%

The answer, of course, is that the direct procedure, as practiced today, in itself inadequate You get a very good result if you relieve the steno[illegible] completely, but at the same time you leave the ventricular septal defect, a[illegible] these patients pass from the clinical state of pulmonic stenosis into the clini[illegible] state of ventricular septal defect, and therefore they become a problem closure of the ventricular septal defect.

I think the correct treatment for tetralogy must concern itself in relief the pulmonic stenosis and in relief of the ventricular septal defect. That what we have to progress toward, and I go back to my opening remark—th we must view this thing with an historical background. The anastomot operations help us in no way in this, whereas the direct operations do pave t[illegible] way toward a complete cure of the condition

DR POTTS

Dr. Charles Dubost, of France, will continue with a discussion of retrogra[illegible] valvotomy in pulmonic stenosis

DR. DUBOST

The retrograde approach for orificial pulmonic stenosis seems to us to be an interesting technical possibility for certain cases of Fallot's tetralogy, of trilogy or of pure pulmonic stenosis Although the objective sought is the same as that obtained by the operation by the ventricular method, as described by Sir Russell Brock, our opinion is that in certain cases the retrograde method has precise indications

We have, in fact, noticed in a series of valvotomies carried out by the ventricular method that there was, in one patient, a rupture of the ventricle wall which could not be sutured, and in another, a secondary aneurysm of the sutured ventricle. As a result of these accidents, we have thought that it would be advisable to carry out the operation of valvotomy by way of the left pulmonary artery



Figs 1 and 2 Secondary aneurysm after transventricular valvotomy

The left pulmonary artery and its branches are liberated and isolated by tapes The tape which encloses the trunk of the artery is passed through a Rumel-Belmont tourniquet. The pericardium is opened and the orificial valvular character of the stenosis is examined and checked, and the pressures are measured.

Then, a little opening is made in the side of the pulmonary artery, into which is inserted a valvulotome with two receding blades This instrument is guided through the stenosed orifice and its blades are opened in successive stages up to a maximum opening of 2 cm. The instrument is then withdrawn and replaced by a two-way dilator which is expanded to complete the opening of the valve. The arteriotomy is then sutured and the tapes are released The pressure is taken again both in the ventricle and in the pulmonary artery

This operational procedure is certainly not perfect. The operation is a longer one than that carried out by Brock. We think, with Brock, that in the case of patients who are suffering greatly from cyanosis, and in the case of

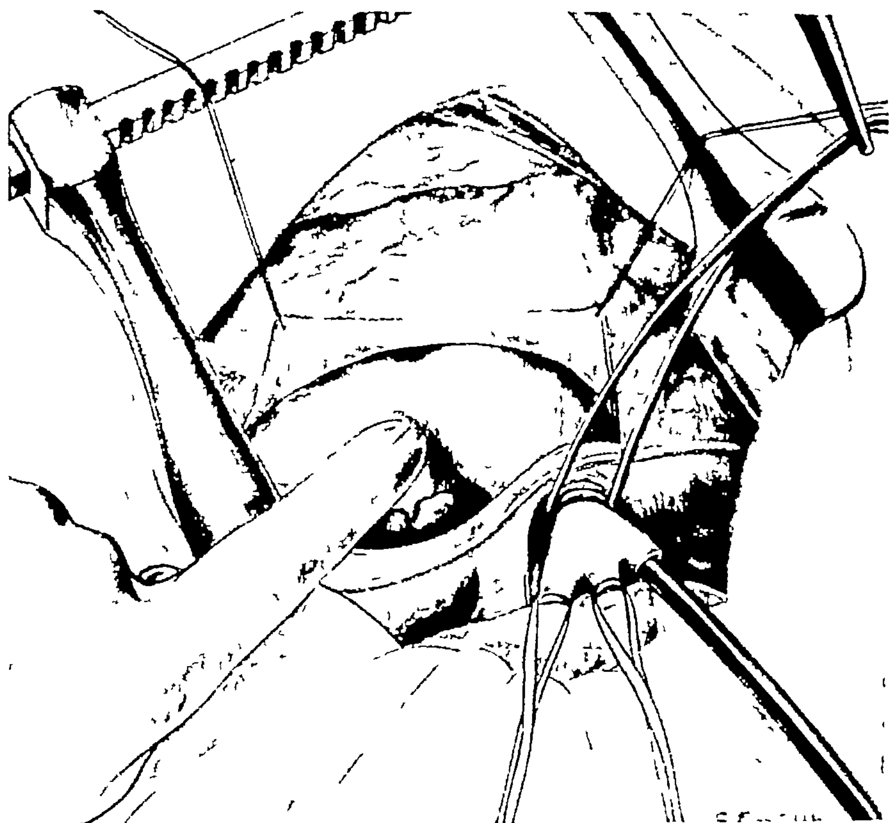
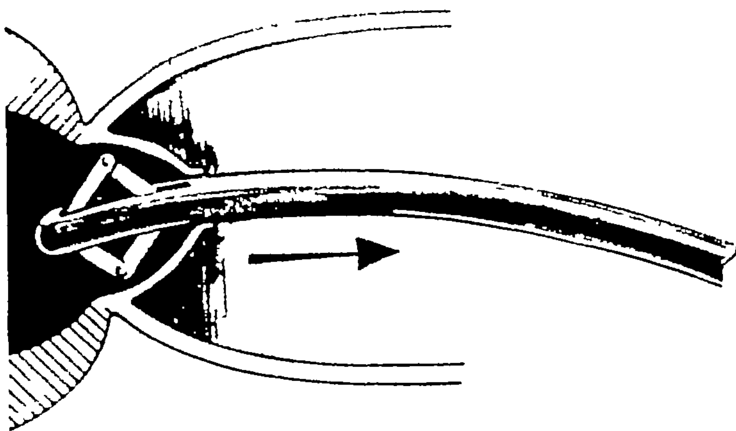


Fig 3 The pulmonary valvulotome is introduced through the valve



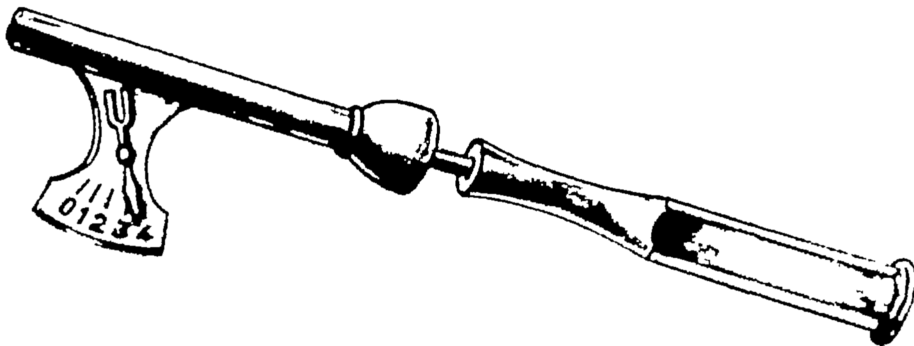


Fig 4 The blades are then opened and the instrument is withdrawn.

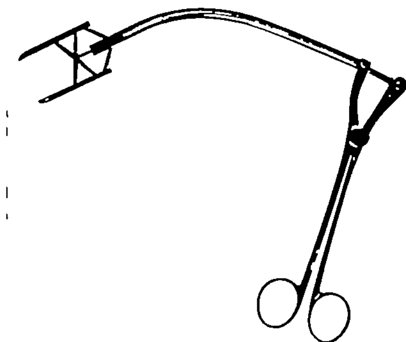


Fig. 5 Pulmonary dilator

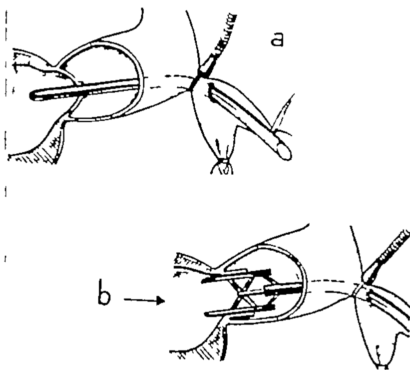


Fig. 6 The valvulotome (*a*) is replaced with the dilator (*b*) which is widely opened.

babies, it is preferable to act quickly and to adopt the transventricular method. On the other hand, the retrograde method is easily carried out, without haste and without any remarkable loss of blood, in both children and adults.

TABLE 1 SURGERY IN CONGENITAL HEART DISEASES WITH PULMONARY STENOSIS
(1948–1954)

450 CASES	Blalock-Taussig's operation	380
	Potts' operation	13
	Pulmonary valvulotomy	57

TABLE 2 VALVOTOMY FOR PULMONARY STENOSIS
57 CASES

Disease	*Transventricular*		*Transpulmonary*	
	Cases	Mortality	Cases	Mortality
Tetralogy	2	2 { 1 V fibrillation; 1 hemorrhage (fibrinolysis)	16	2 { 1 postop hemorrhage; 1 c standstill
Trilogy	5	1 { 1 c standstill (postop hemorrhage)	16	2 { 1 V fibrillation; 1 postop. hemorrhage
Pure pulmonary stenosis	6	2 { 1 rupture right V wall; 1 c standstill (48 hours)	12	1 1 V fibrillation
TOTAL	13	5	44	5

The operation has been used rarely in Fallot's tetralogy, because in 450 operations carried out for congenital stenosis, we have found only 18 cases of pure valvular stenosis.

In Fallot's trilogy and in pure pulmonic stenosis, the results we have obtained are satisfactory, at the price of a 10 per cent mortality (3 deaths out of 28 cases treated).

The results obtained would seem comparable to those which Brock, Bailey, Potts, and others report after ventricular valvulotomy.

DR SWAN

I am going to confine my brief remarks to the pure pulmonary or isolated valvular lesion, and will discuss the reasons which led us to search for an operative procedure by which we could attack this lesion under direct vision.

We thought it was a good idea to study the patient with objective evidence before and aftery surgery, in order to attempt to evaluate our results. We were disappointed in some of our results. In 1953 we tried to find what we could from the literature in this regard (Table 1). These are all of the published data that we could find at that time Note the right ventricular pres-

TABLE 1 PHYSIOLOGIC OBSERVATIONS BEFORE AND AFTER TRANSVENTRICULAR PULMONARY VALVOTOMY

	Preoperative		*Postoperative*			
Author	R. V Pressure	P A. Pressure	R. V Pressure	P A Pressure	Per cent reduction R. V Pressure	Residual stenosis R. V P A. (Systolic)
Lurie and Shumacker	120/0	2/-2	101/0	3/-2	21 0	98
	65/-7	16/4	39/-5	18/6	74 0	21
	82/-6	46/5	44/-6	26/11	73 0	18
	105/4	46/13	106/0	33/12	-1 0	73
	55/0	19/3	48/6	20/9	28 0	28
	127/-7	5/-8	84/-3	14/4	44 0	70
	142/2	17/5	122/-7	14/5	18 0	108
Bing et al.	165/36		135/39		22.0	
	122/30		100/3		24.0	
	84/60		37/14	20/13	87 0	17
	126/40		92/34		35.0	
	145/3	24/9	111/5	35/12	30 0	76
	118/13	22/9	90/10		32 0	
	166/26		72/8	20/9	70 0	52
	128/0	30/17	38/2	26/11	92.0	12
	90/0		98/12		-13 0	
Kirklin	196/5	11/8	65/8	16/12	79 0	49
	55/5	18/10	40/5	20/12	60 0	20
Galligan et al.	260/0		60/0		87 0	
	99/11		58/6		59 0	
Humphreys et al	217/9		69/2		79 0	
	162/12		78/12		64 0	
	201/16		77/13		73 0	
	100/0*		40/0*	77/4	33 0	
Blount et al.	200/5	16/20	151/1	22/13	29 0	129
	166/-7	22/13	38/2	25/10	94 0	13
	221/10	18/11	60/6	35/10	84 0	25
	142/-1	13/6	34/-5	14/8	96 0	20
	110/3	16/7	40/2	14/9	88 0	26

* Obtained at the operating table
From Journal of Thoracic Surgery

sures before and after operation. In all of these groups, great variability was noticeable.

Dr Keith has demonstrated an example of one of these valves They are often very fibrous and stiff, and they will slide over the blade of a knife. Therefore, we thought that operation under direct vision might be a better operation if it could be done safely

With the development of hypothermia a technique was available to explore this possibility, and an operative method was therefore devised (Figs 1 and

2). After occlusion of the venae cavae, the pulmonary artery is clamped distally so there will be no back bleeding, and an opening is made to expose the conical valve It is then possible to make one, two or three incisions through the valve all the way to the valve ring, thus creating an opening as large as the ring or, in other words, as large as possible.

This can very easily be done deliberately in a period of two and a half to three minutes. In fact, it can be done without hypothermia, but I think the

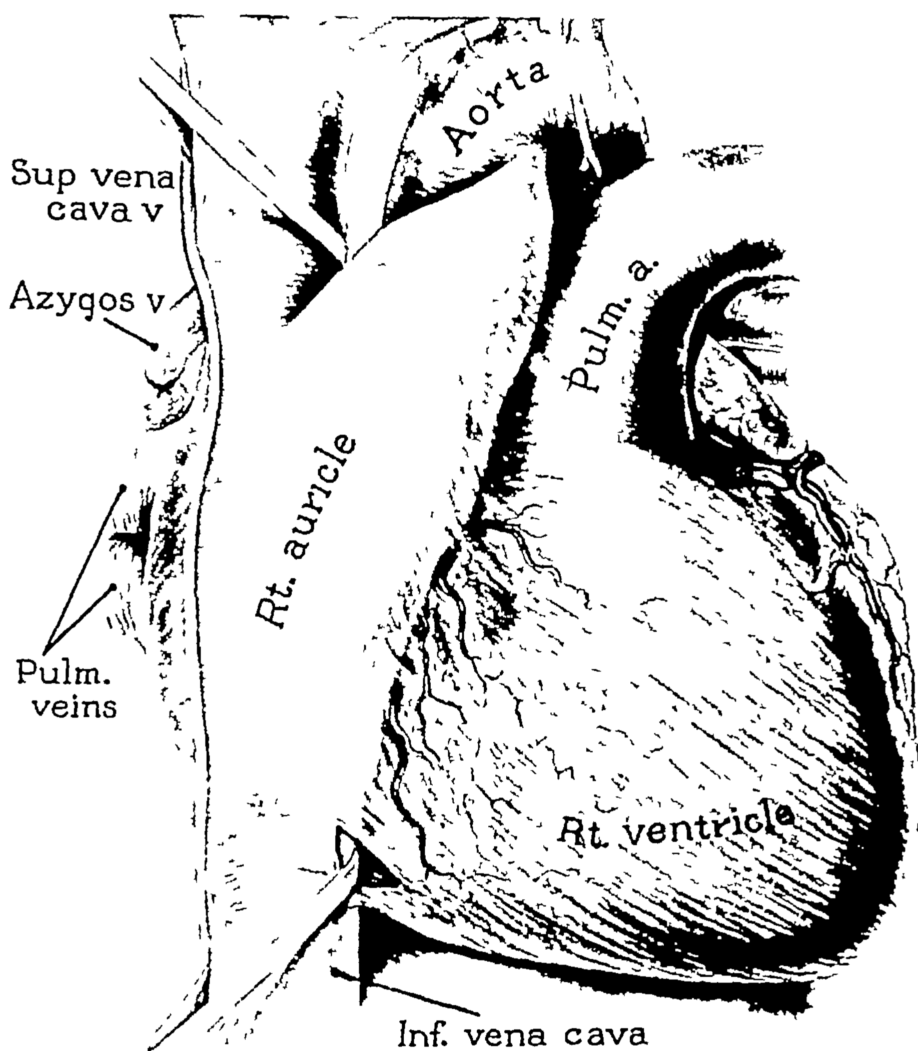


Fig. 1 Ligation of azygos vein and application of tapes to superior and inferior venae cavae preparatory to exposure of the pulmonary valve (subject under hypothermia)

factor of safety and the sense of hurry, if one had only three minutes to do the operation, would be undesirable.

Just before closure the finger is placed through the valve and palpation of the infundibulum is made, to be sure that an associated infundibular stenosis is not being missed The heart is then filled with saline solution and the artery closed at leisure

The results of this maneuver are typified by the pre- and postoperative tracing of the pullback of the catheter from the pulmonary artery to the right ventricle (Fig 3).

In our first 5 patients, who were studied carefully, we had the right ventricular pressures before and after operation (Table 2) You will see that in all instances except one there is less than 10 mm of mercury gradient across the valve.

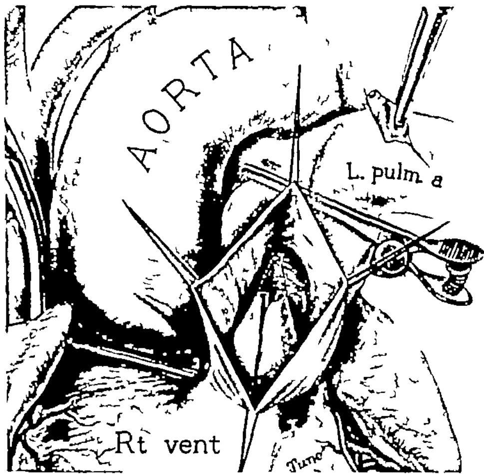


Fig 2. *Pulmonary valvotomy under direct vision Note that the aorta is also occluded.*

This type of study has been continued by my colleague, Dr Blount. All of these patients have been studied postoperatively, and this type of result is seen uniformly We have had 12 cases and have had no deaths

TABLE 2 RESULTS OF PULMONIC VALVOTOMY USING PULMONARY ARTERY APPROACH
(Pressure· mm. Hg)

Patient	*Pre-op*		*Post-op*		*Decrease rt vent pressure*	*Decrease rt vent pressure*
	R. V	P A.	R. V	P A.	(mm. Hg)	/
R. H.	93/3	23/12	29/0	23/8	64	100
J K.	142/–2	17/7	19/–5	15/3	123	100
G S	110/3	18/12	32/–1	32/9	78	98
M. G	119/–5	18/9	29/7	25/9	89	100
D M.	115/54	—	29/7	15/9	86	100
Average	116/11	19/10	28/1	23/7	88	100

From Journal of Thoracic Surgery

M.G Pre-operatively (8-4-53
R.V = 119/-5
P.A. = 18/9
Patient at rest

M.G Post-operatively (3-8-54)
R.V = 29/7
P.A = 25/9
Patient exercising

Fig 3 *Upper record* Tracing of pressures in cardiac catheter as it was pulled from pulmonary artery through stenotic valve into right ventricle. The differential in pressure is striking *Lower record* Pressure tracing from same procedure following relief of pulmonic stenosis by valvotomy under direct vision. The systolic peaks of right ventricle and pulmonary artery are almost identical in height.



Question· In evaluating the operative results of the direct versus the indirect method, did you compare patients falling in similar age groups? *Answer* (*Sir Russell Brock*)· That is not possible to analyze, except that I imagine what is in back of the questioner's mind is whether they included considerable variations in age, such as have been shown—that is, under the age of 3 or 4 years.

We all know that the operative mortality and the results are liable to be muc ct young children The answer is that in the on ost all of them were over the age of 4, in t e I have not had the experience that Dr. se 'oung children.

ion: after the indirect on p

Answer (Sir Russell Brock) I can t give you the exact number, but I would say it is about fifteen. I can tell you that the numbers are increasing steadily

I have actually operated recently on 2 of the 10 patients whom Dr Blalock very kindly operated on in 1949 The thing that worries me, apropos of this, is that recently I operated on a patient on whom a well known surgeon had done an aortic-pulmonary anastomosis The child s condition was totally unsatisfactory, and the surgeon could offer no alternative. The direct operation was quite simple.

Question Do you advise operation for pure pulmonic stenosis when symptoms are minimal and the right ventricular pressure is about 65 mm of mercury? I think it would be well if Dr Swan added to that what his indications are. When does he do an operation for pure pulmonic stenosis?

Answer (Dr Swan) I would think the number selected is a very good number to start an argument about. I do not think 65 mm of mercury pressure in the right ventricle would lead us to operate on a patient if the patient were asymptomatic and if the heart did not show a right ventricular strain pattern However, the same level of pressure in a patient who did have symptoms or was cyanotic or associated with a tetralogy, would lead us to operate on that patient.

In the high pressure levels, that is 70 mm. of mercury or more, we feel that, although it is known that pressures of this magnitude can be tolerated for a long time, nonetheless eventually most of these people will get into trouble, for this reason we think it desirable to restore their pressure relations essentially to normal.

Question What technique will you use to deal with an infundibulum from above through the pulmonary artery?

Answer (Dr Swan) I believe I am fortunate that I have not had to make that decision, but we are prepared to deal with it by attacking the infundibular stenosis by its removal through the hole in the valve. I believe it would be quite possible to resect bites of the infundibulum and enlarge it with a knife until it is possible to make the opening in the infundibulum as large as the ring of the valve. If it were very low, this might be difficult.

On the other hand, if infundibular stenosis is present it is my opinion that one can diagnose this by examining the heart. If the diagnosis is made before the pulmonary artery is opened (as I think it should be), then I believe one would approach the infundibular stenosis directly by the myocardial incision, and perhaps then one would have the problem of being sure the valve above it was not stenosed, if it were, I think I would attempt to use instrumentation by direct vision from below, rather than by direct vision from above.

Question: How did you pull out the air in the ventricle after the valvulotomy?
Answer (Dr. Swan): I didn't pull it out, I allowed it to rise to the top of a fluid level, and the fluid level is obtained by pouring a large quantity of Ringer's solution into the heart and allowing the water to displace the air in the chamber of the heart

The important point here, of course, is that the incision in the heart must be the uppermost portion of the operative field, in order that no pockets form within the chamber, and in order that the air will all be excluded by this method Fortunately we have not had any trouble in the pulmonary valvular group with coronary air embolus

Question: What level of pulmonary pressure do you consider an indication for operation?
Answer (Dr. Swan): In the presence of symptomatology we would operate at under 70 or 75, but in the absence of symptomatology I think a pressure of 75 or greater would lead to operation

Question: Sir Russell, at what pressure would you operate?
Answer (Sir Russell Brock): I would not disagree with what Dr. Swan has said.

Question: Why are so many children very well years after shunt operations, while the chief objection against shunt operation is that the strain on the right heart still exists?
Answer (Dr. Potts). We have found that in the electrocardiographic studies of many patients the right heart strain disappears. Some of them have a combined heart strain, but over a period of months the right preponderance disappears

Question: How large an opening would you make in a 17-month-old cyanotic child weighing about 20 pounds with hematocrit of 74 per cent?
Answer (Dr Potts): In little children we make the incisions in the pulmonary and in the aorta 5 mm long instead of 6 mm, as in the average child A 5 mm incision will give approximately 3 mm. in diameter opening. We operated on one child two weeks old who weighed 4 pounds, 13 ounces, and made such an opening Fortunately, the child recovered One year later it died of meningitis, and at post-mortem examination there was an opening 3 mm. in diameter, and the child had remained pink and had kept its continuous murmur.

Question: Please comment on the life history of patients with isolated pulmonic stenosis What is the life expectancy? Should they all be operated on?
Answer (Dr. Taussig) I think we have all agreed that not all patients with pure pulmonic stenosis should be operated on Some are very mild, and if we catheterize all patients I think we will find there are

more extremely mild patients in whom symptoms never develop, and who do well over a period of years

I am sure that the life history depends upon the severity of the pulmonic stenosis In all malformations there is great difference in the severity of the abnormality If severe pulmonic stenosis with high pressure and right ventricular strain leads to progressive enlargement, then certainly operation is indicated, and is of great benefit

Question Do you expect a 3 mm. opening to be adequate at the age of 10, 15, 20 or 25? Have you reoperated on such individuals and made the opening larger?

Answer (*Dr Potts*) We have not reoperated, but you will remember we did some experiments on growing pigs, operating on them when they weighed 25 pounds and killing them when they weighed 250 pounds We found that the opening had practically doubled in size in some of them

We think the opening does enlarge somewhat, but the few that I have done, who have come back to us, have not had much enlargement over a period of a couple of years There is some growth, but not a great deal

DISCUSSION

Dr Potts

I think it would be very appropriate if Dr Taussig would open the discussion on the treatment of the tetralogy of Fallot.

Dr Taussig

In the first place, I think we would all agree that in anything, the more methods of approach we have, the better off we are. The more alternatives that are possible for the surgeon to deal with in a situation, the better

I think Dr Blalock would agree with Sir Russell, that the ideal would be a complete repair of the defect. He has said that he felt that sometimes the overriding of the aorta is so marked that it would be very difficult to completely repair the defect and be sure that it would hold.

I also would add that in some instances the pulmonary artery is too small—so that even if you cut out the interior, it would not be possible to completely restore the heart to normal. Therefore, I think you will probably always find a place for an anastomosis, and it would be only when the repair of ventricular defects is safe and the mortality rate is low that we will be able to combine the two procedures of cutting out the obstructing area and closing the ventricular defect.

The long term results remain to be seen in both groups, and whether you are going to weaken the heart muscle, and whether that upper chamber is strong enough to take the increase over a period of years, is another question.

There is one other line of thought that I would like to leave with you.

I believe we all agree that in valvular pulmonic stenosis with an intact septum, valvotomy is the operation. I leave it to the surgeons to discuss the types, but we haven't mentioned at all today one thing which we should consider, and that is that there are a certain number of patients with a single ventricle and pulmonic stenosis, which may be taken for tetralogy of Fallot.

If there is a single ventricle with pulmonic stenosis, I think it would be a sad mistake to do a valvotomy or at least a complete valvotomy, and try to remove the pressure gradient when you have systemic pressure in the right ventricle, because then you would be injecting blood to the lungs under systemic pressure, and that certainly would not be desirable.

Sir Russell Brock

First of all, I would like to say that I agree absolutely with Dr Helen Taussig in her statement that there must be cases in which the outflow tract of the right ventricle is too small to make it desirable to do a direct operation, having particular regard to the inevitable period of reactionary swelling and edema that must occur after any direct surgical operation.

There are cases, especially in young children, in which an anastomosis may be desirable That is a question of surgical judgment and selection.

On the other hand, I would point out that if the pulmonary outflow tract is not used functionally, then it can never grow, and one of the great advantages of the direct operation is that if it is done in a young patient it will give the outflow tract a possibility of developing and growing with the rest of the body.

Victor Bernhard (*Chicago*)

We have had the opportunity to review the catheterization data on a series of patients with infundibular stenosis, and have been able to differentiate on the basis of the pressure curves two distinct types, one of them being the sclerotic variety, wherein the right ventricular and infundibular pressures arise simultaneously but with a gradient between them, as in any kind of stenosis In the other type the right ventricular pressure rises along with the infundibular pressure for about 0.1 second, at which time the pressure in the infundibulum begins to fall, while the right ventricular pressure continues to rise to a new height.

We interpret this as a method for differentiating the sclerotic type from that which is apparently due to the contraction of the subinfundibular ring during systole.

Tyge Sondergaard (*Aarhus, Denmark*)

I should like briefly to demonstrate my technique of transarterial valvotomy performed through the main stem of the artery

It is a rather general impression at present that the results of standard valvotomy in isolated pulmonary stenosis are not quite satisfactory. The drop in pressure is not uniformly good, and it is obvious that Swan and his group have found one solution to this problem

Our group in Denmark has been working for only two years, and we have a very small number of patients, but I have used a technique which I developed a few hundred miles from here—in Buffalo, in 1948—and we have found no reason to consider any change.

I use a double ring clamp with one ring covered with a piece of rubber tubing (Fig 1) Figure 2 shows how it works—on a little piece of rubber with an incision The lower ring is inserted through the incision, and the incision is grasped between the two rings, so it is completely encircled.

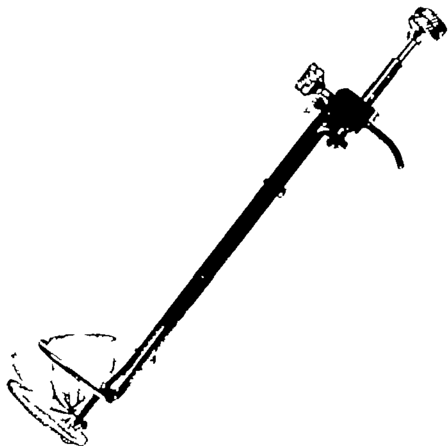


Fig 1 Double ring clamp used in transarterial valvotomy

When you cut the ligature around the rubber tube down below, you have established an operating tunnel You can do it in a matter of seconds without any blood loss, and you can work inside the artery for as long as you wish.

Figure 3 illustrates an operation performed a couple of years ago You see the ring clamp in place on the dilated pulmonary artery I clamped a longitudinal fold of the pulmonary artery with a soft clamp and opened the artery with an appropriate incision, slipped in the lower ring, clamped on the upper ring, and took off the soft clamp The circulation goes on all during the procedure, unhindered. I use a tourniquet around the rubber tube, and insert the cutting and dilating instruments (Fig 4) We have made some dilators which dilate up to 35 mm. With this technique we have all the time we need There is no blood loss, and you can dilate much more efficiently than when you

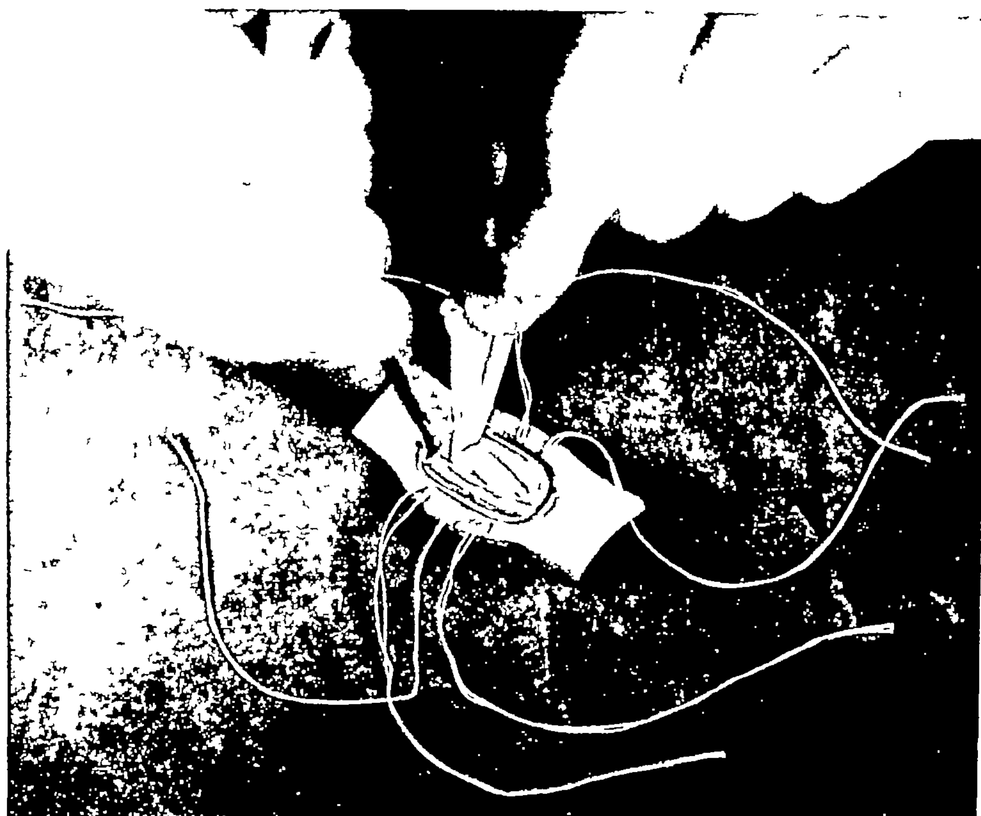


Fig 2 Use of clamp demonstrated on a small piece of rubber with an incision.

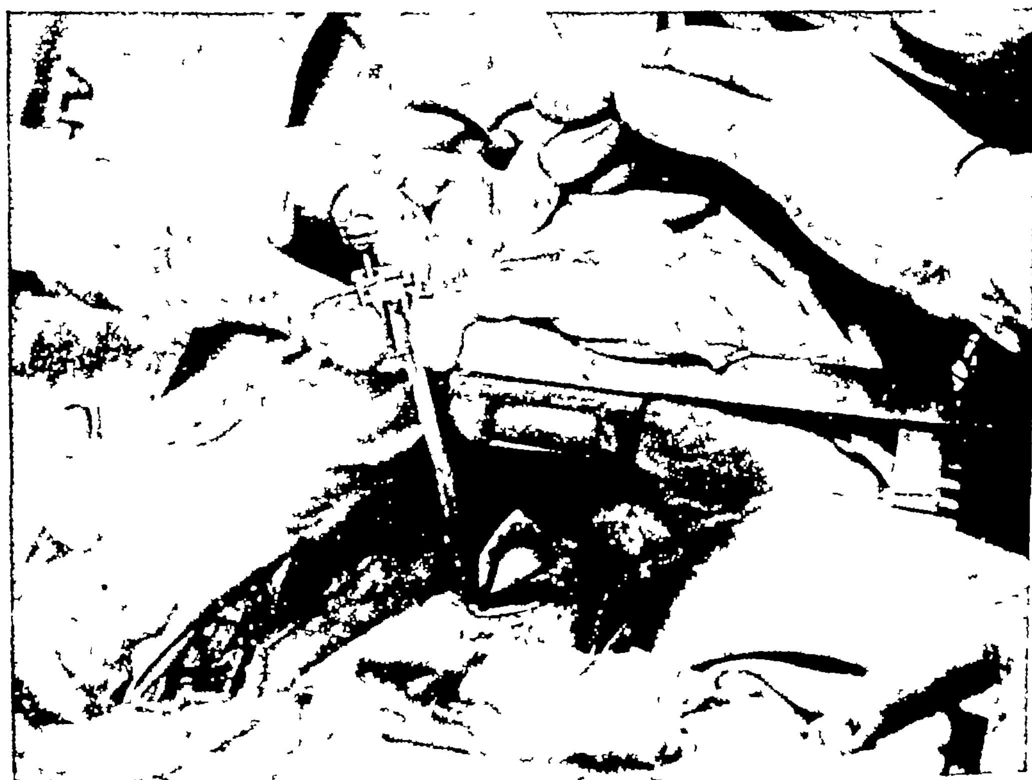


Fig 3 The ring clamp in use on the dilated pulmonary artery

push the instruments up against the valve. In this case you pull the instrument through the valve. It gives a better control, at least in my opinion

We have had no mortality We have had postoperative catheterization in only 6 patients The results have been so good that I considered it worthwhile to report it briefly today The average drop in pressure has been 120,



Fig 4 The my clamp with a tourniquet around the rubber tube.

and the pressure gradient before the operation was 150 The average pressure gradient after the operation was 21 mm., varying from 14 to 36 mm.

You cannot avoid a pressure gradient, even if you split the valves completely, because these patients have a dilated pulmonary artery, where the valve ring is the narrow point, and you have to have a drop in pressure there. Besides that, many of the patients in this group had an atrial septal defect as well, with a flow from left to right, which alone gives a functional pressure gradient via the pulmonary valvular ring.

ADJUSTMENTS BETWEEN THE SYSTEMIC AND PULMONARY CIRCULATIONS

RICHARD J BING (*Birmingham*)—CHAIRMAN

CLINICO-ANATOMIC CORRELATIONS

J. FRANCIS DAMMANN, JR. (*Charlottesville, Virginia*) AND
CHARLOTTE FERENCZ (*Baltimore*)

Interest in the pathologic physiology of congenital heart disease centered mainly around the exact structural defect within the heart until Edwards and his associates[1–3] directed attention to the lungs and the important role of the pulmonary vascular bed in governing the clinical course of congenital heart disease Changes in the pulmonary vascular bed in association with various cardiac anomalies have been described by many authors[4–29] but there appear to be widely divergent views as to their etiology The lack of correlation between the degree of pulmonary vascular change and the size of the cardiac defect, the occasional presence of thrombotic lesions, and the occurrence of severe pulmonary vascular change in the *absence* of congenital heart disease, are most frequently cited as evidence of an independent, "idiopathic" etiologic mechanism [30] While there is no doubt that "primary" pulmonary hypertension represents a definite entity,[31–35] there is a growing body of evidence to support Edwards' concept[36–46] that in association with congenital malformations of the heart, changes in the pulmonary vessels are not necessarily a separate anomaly. Such changes represent a compensatory mechanism which enables the patient to exist However, these changes, at first compensatory, progress and become detrimental to the patient

This study of the anatomic alterations of the pulmonary vascular bed was undertaken with a triple purpose To clarify the interrelation of pulmonary vascular changes with the type of malformation of the heart present, to examine this relationship in various age groups and clinical syndromes, and, by improving our understanding of the course of the disease, to aid in the rational application of corrective surgical procedures.

MATERIAL

Over the past four years lung sections were collected from autopsy and surgical biopsy material from more than 200 patients with congenital heart disease, and from more than 100 "normal" patients of all ages, who died of causes unrelated to the heart and lungs.

Elastic tissue and hematoxylin-and-eosin stains were made An arbitrary method was set up for the evaluation of the pulmonary vascular changes. Small muscular pulmonary arteries associated with or lying clearly apart from small bronchioles were selected The total diameter of a vessel cut end-on was measured The thickness of each layer, adventitia, media and intima was

recorded. Arbitrarily, the diameter of the lumen was divided by twice the thickness of the media and intima added together, thus giving a ratio of lumen size to wall thickness Ten vessels were measured and their ratios averaged. Because this method of evaluation is subject to marked error, every effort was made to avoid subjective reading Most of the sections from normal patients, as well as a large number of abnormals, were sent from the Children s Memorial Hospital, Montreal, through the courtesy of Dr F W Wiglesworth. At the time these were received, the only accompanying information was the age of each patient. It was gratifying to note that the overwhelming majority of the normals were selected correctly by the examiner and that, among the abnormals, a correct general differential diagnosis based on high pressure, high blood flow or venous obstruction was made in about 80 per cent.

The malformations were grouped according to the predominant type of strain placed upon the lungs Three main groups were established

I Defects between the ventricles or great vessels in which both increased pressure and blood flow may act upon the lungs and in which there is a common ejectile force.

II Defects in which a high pulmonary blood flow is the chief physiologic change.

III Lesions of the left side of the heart causing an obstruction of the pulmonary venous return.

In this paper we will deal with the first two groups alone. Graphs were constructed to correlate the microscopic and clinical data from each group The lumen wall ratios were plotted against the age of the patient Semilogarithmic paper was used to allow for a wider spread of data in the younger age groups The clinical and laboratory data of each case were analyzed independently and indicated by special symbols In this way the relationship between pulmonary vascular changes and the clinical picture at various ages becomes apparent at once.

NORMAL LUNGS

Before discussing abnormal pulmonary vascular changes, it is important to consider the normal evolution of the pulmonary vasculature. Studies have shown that at birth the structure of the small intrapulmonary arteries resembles that of the small arteries and arterioles of the systemic circulation.[47] Progressive changes occur in the pulmonary arteries during the early months of postnatal life. Figure 1 is a photograph of a typical small muscular artery of a newborn infant. The lumen is small, frequently completely closed, the intima is thin. The media is thick, as is the adventitia. This vessel appears similar to a systemic vessel. At 10 weeks of age (Fig 2) a definite change has taken place. The lumen is relatively and absolutely larger and the media has thinned out. At 2½ years of age (Fig 3) further thinning out of the media and widening of the lumen have taken place. Except for the presence of the elastica interna and externa, often difficult to discern, the small pulmonary arteries look like small veins These changes are clearly demonstrated by the application of the lumen wall ratio (Fig 4) At birth and during the first 6 months

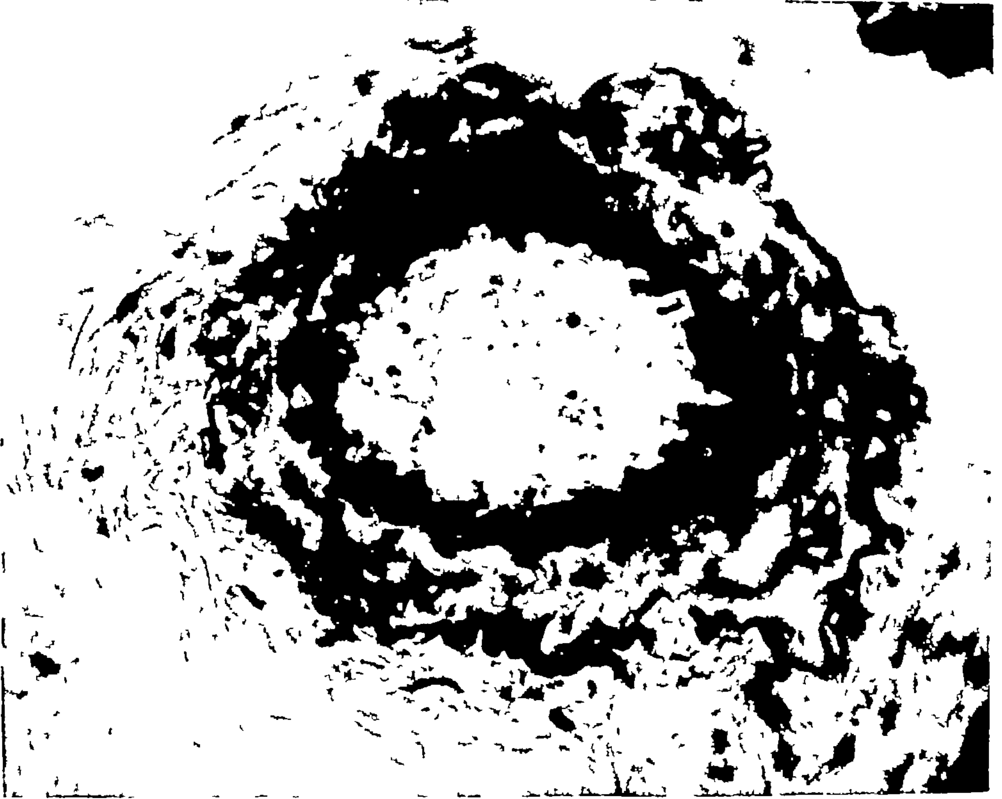


Fig 1 Small muscular pulmonary artery of newborn infant

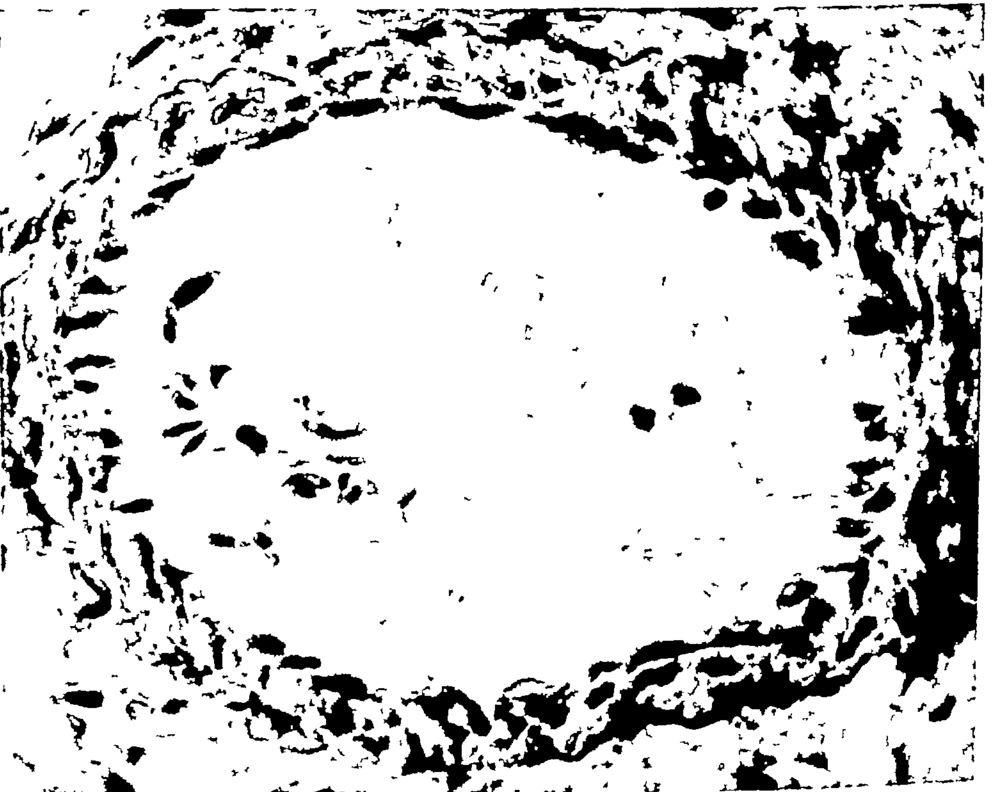


Fig 2 Small muscular pulmonary artery of a 10-week-old infant

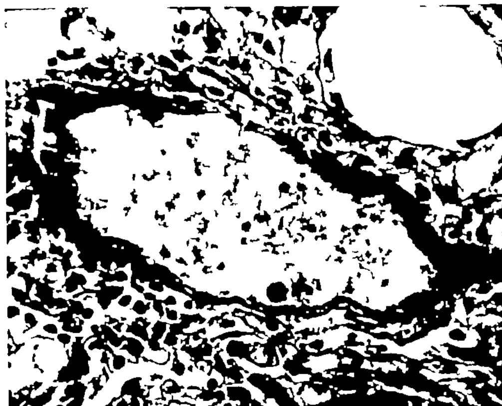


Fig 3 Small muscular pulmonary artery of a 2½ year-old child

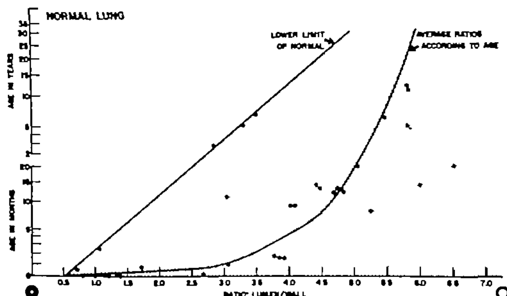


Fig 4 Lumen wall ratio—normal patients.

of life many normal patients had ratios below 2.5 but after 6 months there were only a few Furthermore, although all of the older patients had high ratios, the age at which this appeared was variable. Thus, some patients at 6 months of age had ratios of 5 0, whereas others did not reach this ratio in 6 years

The physiologic effect of this progressive increase in lumen size and decrease in wall thickness is a progressive fall in pulmonary vascular resist-

ance,[48-52] so that in the normal individual, despite equal volumes of blood flow, a pulmonary mean pressure 20 to 25 per cent of the systemic mean pressure suffices to propel the blood through the lungs.

With this analysis of the lumen : wall ratios in normal children as a framework of reference, deviations present in cases of congenital heart disease may be evaluated

ABNORMAL VASCULATURE

GROUP I Defects between the ventricles or great vessels in which both increased pressure and blood flow may act upon the lungs and in which there is a common ejectile force.

When the cross-sectional area of a ventricular septal defect approximates that of the aortic orifice, the systolic pressures generated in each ventricle must be the same. In Edwards' terminology a "systemic right ventricle" or "common ejectile force" is present. Systolic pressures in the aorta and pulmonary artery being equal, the distribution of blood into the two circulations will be dependent upon their respective peripheral vascular resistances Furthermore, in contrast to the normal condition in which the lungs can accept a four-fold increase in blood flow without a change in pressure, when a common ejectile force is present the lungs are filled to capacity. That is, an increase in blood flow cannot occur without an increase in systemic and pulmonary artery pressure or a decrease in pulmonary vascular resistance. Pulmonary vascular resistance is not static but, rather, is dependent upon what evolutionary pattern the small pulmonary arteries follow. Theoretically, there are three possible courses that the pulmonary vascular bed may follow after birth in the presence of a common ejectile force.[53-54] These result in three distinctly different clinical syndromes or phases

Phase I. Pulmonary vascular evolution follows a relatively normal course with falling peripheral resistance in the pulmonary vascular bed. Consequently, a progressively larger proportion of the cardiac output passes to the lungs and a relatively smaller quantity of blood to the body. In order to maintain an adequate systemic blood flow the cardiac output must rise High output failure frequently ensues The clinical picture is that of an underdeveloped, thin child with a large heart, excessive blood flow to the lungs and high output cardiac failure

Phase II The fetal state of thick-walled, small-lumened vessels and high pulmonary vascular resistance continues with approximately equal peripheral resistances in the two circulations Thus, blood flows will be similar and a state of circulatory balance will exist, allowing normal growth and development The patient may be symptom-free, cyanosis is minimal or absent, no significant cardiac enlargement occurs

Phase III The pulmonary vascular resistance increases gradually with the development of secondary intimal changes and eventually exceeds the systemic resistance More blood goes to the body than to the lungs Growth and development are retarded owing to anoxia, dyspnea and cyanosis are present.

In each of the three phases the underlying cardiac malformation may or

may not be the same The patient may have a large ventricular defect, single ventricle, true truncus arteriosus, large patent ductus arteriosus or aortic septal defect. The common denominator in all phases, no matter what form of malformation exists within the heart, is the presence of a common systolic pressure in both right and left ventricle, pulmonary artery and aorta.

Figure 5 depicts the results of an analysis of 53 patients with ventricular septal defects, single ventricle, patent ductus arteriosus or aortic septal defect. In each instance a proven common ejectile force was present (i.e., the systemic and pulmonary artery pressures were measured and were found to be equal) Each case was allocated to the appropriate phase on the basis of clinical and laboratory data independently of the result of the microscopic examination of the lungs A closed circle was used to designate Phase I, a square, Phase II, and a triangle, Phase III

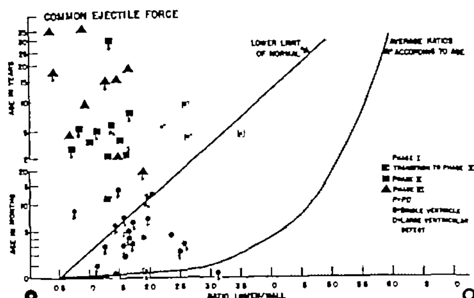


Fig. 5 Lumen wall ratio of patients with a common ejectile force.

It is readily apparent that the lumen wall ratio of these patients deviates strikingly from the normal. There is a pattern to the deviation, however Patients presenting the clinical picture of high output cardiac failure (Phase I) tended to be young and had ratios somewhat lower than normal patients of the same age but larger than the newborn. Patients with a balanced circulation (Phase II) ranged in age from 6 months to 10 years and had definitely reduced ratios Cyanotic patients in Clinical Phase III were older and had ratios markedly below those of normal patients of their own age. The vascular progression, then, is for a small pulmonary artery, normal at birth, to become larger lumened and thinner-walled during the first weeks of life but to a lesser degree than in the normal patient. Following this, there is a gradual shift back toward the thick-walled, small-lumened status and eventually, if the patient survives long enough, vessels have thicker walls and smaller lumens than those found at birth.

There is a clear correlation between this vascular pattern and the progression of clinical signs and symptoms The infant with a large ventricular

septal defect appears normal at birth and for a short time thereaft monary vascular evolution results in a falling pulmonary resistance. Si symptoms of high-output cardiac failure appear, i.e., rapid respiration pulse, pulmonary congestion often diagnosed as or complicated by monia, large heart and large liver. Many of these infants die in co heart failure despite all medical therapy. Patients who survive pa Phase II. One reason for survival is the ability of the heart to hype sufficiently to carry the load A second reason for survival may be a change in the size of the defect as compared to the size of the aortic If the left heart and aortic valve increase in size more rapidly than the a point may be reached at which the defect itself limits the volume c flow which passes through to the lung. Therefore, a pressure gradient between the left and right ventricle, and the course of such a pat

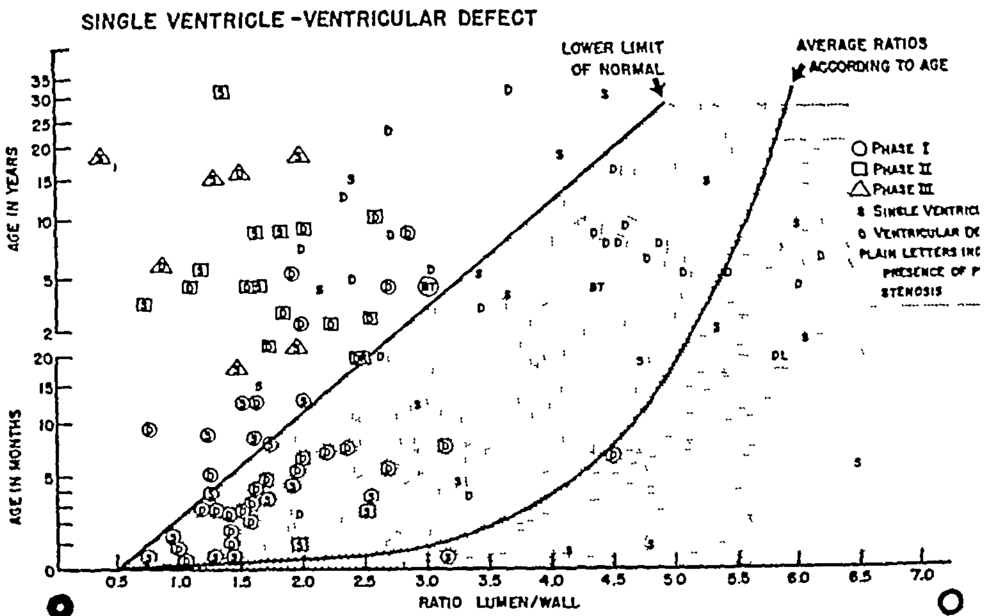


Fig 6 Lumen wall ratio of patients with ventricular defect, single vent

longer is that of a common ejectile force A third reason, perhaps th important, is that normal pulmonary vascular evolution does not co The small arteries regain their thick walls and small lumens and sh absolute increase in muscular thickness and in elastic tissue of the This means that a high pulmonary vascular resistance is regained ar the blood flow to the two circulations is brought within reasonable The clinical status of the patient is improved and his good condition h physician and his family into a false feeling of security and hopes for a future. For reasons that appear to be related to the presence of sy pressure in the pulmonary artery, pulmonary resistance does not rema anced but continues to increase, further medial hypertrophy takes p the small pulmonary arteries and, in addition, intimal thickening is Pulmonary resistance eventually becomes greater than systemic resi Consequently, it now becomes easier for the heart to pump blood in body than into the lungs and the patient becomes cyanotic. This repi Phase III. From Phase I to Phase III, whereas the pulmonary bloo

decreases markedly, the systemic output increases only slightly Systolic pressure changes to only a small degree. Consequently, the total work of the heart is decreased This accounts for the decrease in heart size and the establishment of cardiac compensation

For comparison, Fig 6 depicts the results of an analysis of 100 patients with ventricular septal defects or single ventricle, many of whom had additional pulmonary stenosis. Those patients with a probable common ejectile force were allocated phases according to the available clinical data The plain letters indicate the presence of a clear-cut anatomic pulmonary stenosis There is little deviation from the normal in patients with pulmonary stenosis The lumen wall ratio on the whole tends to follow a normal path. In a few patients with pulmonary atresia low ratios were obtained. This we think is owing to the difficulty of differentiating collateral bronchial vessels from true

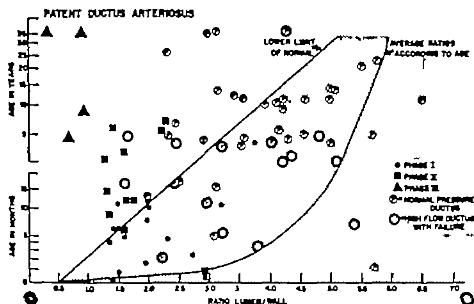


Fig. 7 Lumen wall ratio of patients with patent ductus arteriosus

pulmonary arteries In others among the older age group thrombi similar to those described by Rich[55] were present, causing a lumen wall ratio that was clearly lower than normal.

Figure 7 depicts the results of an analysis of 75 cases of patent ductus arteriosus or aortic septal defect. As in the previous graphs, patients in whom there was objective evidence of a common ejectile force were grouped according to clinical phases Again the pattern of progressive change from Phase I to Phase III is clearly demonstrated. High-output cardiac failure was not uncommon in early infancy Patients with high-output failure had ratios that were somewhat larger than those of normal infants of a comparable age. Patients in Phase II had ratios that were lower than those in Phase I and significantly lower than the normal. Patients in Phase III with a reversal of shunt through the ductus had extreme pulmonary vascular change. In each instance in this series the ductus was huge, being close to the size of the aorta and very short. In each instance systolic pressures in the pulmonary artery and aorta were identical.

In contrast to the patients with a common ejectile force are the patients with a typical small patent ductus arteriosus, a continuous murmur, no elevation of pulmonary pressure and minimal symptoms. The letter "P" designates this group The ratios are well within the range of normal, although they tend to be lower than average ratios according to age. A few in the older age group have shown clear-cut pulmonary vascular change. It is of interest, and we think significant, that one of our older patients with a rather small ratio had an essential hypertension of severe degree; another had a coarctation immediately distal to the ductus. In these patients factors in the systemic circulation tended to increase the volume of shunt through the ductus.

GROUP II. Defects in which a high pulmonary blood flow is the chief physiologic change.

Let us now turn to the second group of malformations, those in which a high pulmonary blood flow represents the basic physiologic change. This group comprises cases of atrial septal defect, anomalous return of the pulmonary veins and communications between the ventricles and great vessels which are small enough so that a pressure gradient is maintained between the systemic and pulmonary circulations. In contrast to malformations in which a common ejectile force exists, the right and left sides of the heart do not function as one

In the presence of a common ejectile force, the lungs play the vital role in determining the clinical course of the patient. In Group II the role of the pulmonary vascular bed is passive The distribution of blood into the pulmonary artery and systemic circulations respectively is not totally governed by the state of the pulmonary vascular bed but is dependent also upon the site and size of the defect and efficiency of the ventricles. The child born with a ventricular septal defect of a moderate size will respond at first as a patient with a common ejectile force, since the ventricular defect approximates the aortic orifice. If the defect is membranous in its site, the size of the ventricle and aortic orifice and the total cardiac output will increase at a faster rate than the ventricular defect. Eventually the point may be reached where the defect itself restricts the volume of blood flow from left to right ventricle As soon as this occurs, the lungs no longer are subjected to a common ejectile force and no longer are filled to capacity. Consequently, pulmonary pressure may drop Furthermore, since the volume of shunt is limited, the shunt cannot increase as total cardiac output increases, relatively more blood is pumped to the systemic circulation, growth improves and the child gradually recovers compensation It is possible that, in a period of time, pulmonary artery pressure may drop to normal and that systemic output may return to normal levels

This sequence of events is followed in the pulmonary vascular bed by a trend toward normal evolution The degree of normalcy is variable, probably dependent upon individual factors as well as the size of the defect and volume of blood flow In some instances, however, and perhaps in all cases of an older age group, there is a gradual decrease in size of the pulmonary vessels and decrease in lumen . wall ratio Whether the etiology of this vascular narrowing is the elevated pulmonary blood flow, repeated pulmonary infections

or pulmonary congestion is not clear As pulmonary vessels become smaller, pulmonary resistance increases and right ventricular and pulmonary artery pressures rise. There is a gradual reduction in shunt since the pressure differential between left and right ventricle decreases When pulmonary resistance has increased sufficiently, the pressure in the right ventricle equals that in the left ventricle and the patient once again has a common ejectile force

Reasoning from the foregoing discussion, it might be expected that the lumen wall ratios of patients with a moderate-sized ventricular defect or moderate patent ductus arteriosus would fail to follow a truly distinctive pattern In Fig 7, patients with moderate-sized patent ductus arteriosus and consequent cardiac failure, high pulmonary blood flow or elevated pulmonary pressure were separated from those with a typical patent ductus and those with a common ejectile force and were designated by a plain "P with a heavy black circle In all age groups there was a marked variation in ratios, with, perhaps, a trend toward the left in the older age group Some are in the common ejectile force zone, others are well within normal limits It seems likely that those patients with a low ratio under the age of 10 are patients who, in infancy, had a common ejectile force. Those patients with normal ratio may represent patients with smaller defects who, at no point, had a common ejectile force. It is important that this difference be clarified, for surgery may be indicated earlier in the group with a low lumen wall ratio

In cases of atrial septal defect, the entire load of the left to right shunt is borne by the right ventricle [56] In early life this shunt is relatively small and increases gradually as the right ventricle and septal defect increase in size. Right ventricular and pulmonary hypertension may occur without any alteration in the magnitude of the left to right shunt. When the right ventricle begins to fail, however, the rise in right atrial pressure will reduce, and even reverse, the shunt through the defect. Thus, in this group, in contrast with Phase III of malformations with a common ejectile force, cyanosis is associated with cardiac enlargement and cardiac failure. The circulatory dynamics of cases with total anomalous drainage of pulmonary veins are similar to those of the atrial septal defects, but the alterations are more severe and pulmonary hypertension and cardiac failure occur early Cases of partial anomalous pulmonary venous return follow the same pattern if the foramen ovale is closed, but if the foramen ovale is patent and allows a right to left shunt to occur, the right ventricular load may be expected to be lessened.

On the basis of these considerations the presence or absence of congestive heart failure was noted in analyzing the data of patients with atrial septal defects (lettered "A" in Fig 8) and those with anomalous venous return (lettered "V") The presence of heart failure is indicated by circled letters The deviation from the normal demonstrated by this group is quite different from that shown by patients with a common ejectile force. The lungs of patients not in failure tend to develop normally From 8 to 20 years of age most atrial defects were associated with vascular ratios at the lower limits of normal. Over 20 years of age a gradual decrease is observed until very low ratios are reached. Two of the oldest patients showed cyanosis indicating a reversal of the shunt. Patients with right heart failure had ratios lower than

those who were compensated. Cases of anomalous venous return showed more pronounced changes and the trend toward abnormally low ratios wa apparentat an earlier age than in the atrial septal defect group. This differenc is in keeping with the pathologic physiology of the two conditions

It would appear, then, that the prolonged increase in pulmonary blood flo is responsible for anatomic changes in the pulmonary vascular bed, but th development of these changes is much slower than that seen in Group . The late appearance of these changes in patients with atrial septal defect i one possible reason why this malformation is classically associated with relatively long life span Furthermore, there is not the same urgency for earl operative intervention as in cases in which a common ejectile force is pres ent.[57] Once, however, vascular changes have narrowed the capacity of th

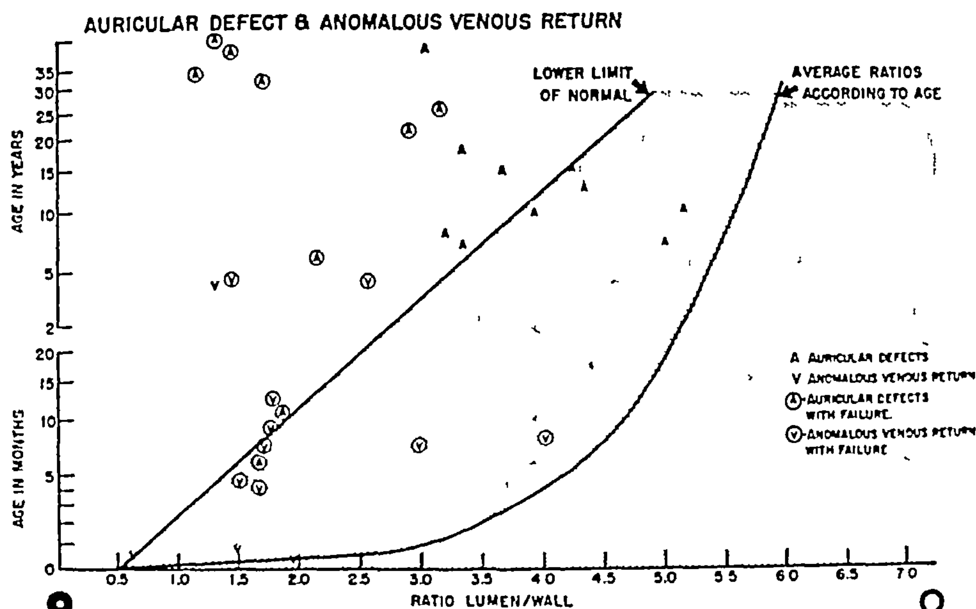


Fig 8 Lumen . wall ratio of patients with atrial defects or anomalies of the venous return

pulmonary vascular bed, the effect on the heart is that of an obstructive lesion. Cardiac dilation and failure supervene and closure of the defect at this stage cannot be expected to alter the course of the disease.

CONCLUSIONS

The findings of this study of the small pulmonary arteries in cases of congenital heart disease and their comparison to those of normal children of similar ages confirm the theoretic concept that changes in the pulmonary vascular bed occur in response to two main influences:

1. A common systemic-pulmonary ejectile force
2. A large pulmonary blood flow

The presence of any one, or a combination of, these factors initiates the gradual and progressive elevation of the pulmonary vascular resistance The clinical picture produced by the malformation of the heart is governed by the state of the pulmonary vascular resistance at any one time Thus, there exists a characteristic life cycle which is dependent upon an excessive, a controlled

or a reduced pulmonary blood flow of malformations with a common ejectile force. The division of the clinical syndromes presented by such malformations into distinct "phases" in the natural history of the disease is supported by the observed anatomic state of the pulmonary vascular bed

When a common ejectile force acts upon the pulmonary vascular bed, its normal evolution from the fetal to the adult state is interfered with. Furthermore, the clinical manifestations of the cardiac defect are dependent on the pulmonary vascular changes In contrast, in the presence of high pulmonary blood flow alone, the lungs play a passive role. The clinical manifestations are dependent not on the lungs, but on the size and site of the cardiac defect and the efficiency of the heart itself Pulmonary vascular changes occur later However, they frequently progress to a point where the clinical picture is again dependent upon the lungs

The progression of pulmonary vascular changes emphasizes the importance of surgical correction of malformations of the heart before irreversible alterations in the small pulmonary arteries render therapy ineffective.

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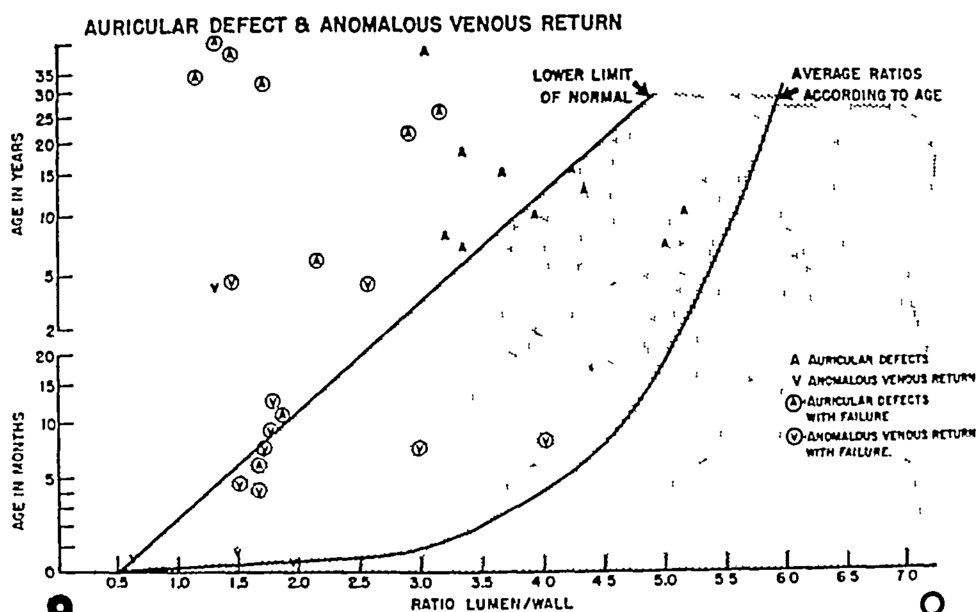


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PATHOLOGIC CONSIDERATIONS IN ADJUSTMENTS BETWEEN THE SYSTEMIC AND PULMONARY CIRCULATIONS

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This presentation will concern itself principally with the pathologic manifestations of a communication between the greater and lesser circulations, either at the level of the ventricles or between the aorta and the lesser circulation. The most common form of the latter is patent ductus arteriosus, which may be considered representative also of a less common type of communication between the aorta and lesser circulation in the form of aorticopulmonary septal defect. The features that pertain to the communications at the ventricular level are essentially like those of patent ductus arteriosus. I shall therefore consider ventricular septal defect and patent ductus arteriosus together. Malformations characterized by a single ventricle will not be mentioned specifically, but remarks about large ventricular septal defect also pertain to single ventricle.[1]

THE NORMAL PULMONARY VASCULAR BED

Since structural alterations in the pulmonary vascular bed will be discussed, it seems appropriate at the outset to define anatomically the components of this bed as they appear in the adult. The elastic arteries accompany the cartilaginous bronchi and branch with these. Arising from the elastic arteries are branches which accompany the noncartilaginous bronchi, these are the large muscular arteries (Fig. 1*a*).

The elastic arteries have histologic patterns similar to that of the aorta and major pulmonary arteries They are characterized by having a media with layers of elastic tissue arranged at regular intervals. There is little intimal tissue other than the lining endothelium and a small amount of supporting collagen. The large muscular arteries characteristically have a well defined medial layer composed of circularly oriented muscle. There is an internal elastic lamina lying between the thin intima and the media, and the external elastic lamina separates the media from the collagenous adventitia.

Arising at right angles from the large muscular arteries are smaller branches (Fig. 1*b*, *c* and *d*). Proximally these vessels have a medial layer composed of muscle with an over-all structure essentially comparable to that of the large muscular arteries (Fig. 1*c*). At this level this branch may be called a small

muscular artery As the small muscular artery proceeds distally, it loses its medial muscle and assumes the structure of an arteriole. At this peripheral level the vessel is lined by endothelium and its wall is composed principally of collagen and a layer of elastic tissue (Fig 1*d*) While it cannot be denied that some muscle fibers may be present at this level, there is no identifiable



Fig. 1 Normal pulmonary vessels *a*, A muscular artery arises as a branch of an elastic artery The former lies to the left the latter to the right (ELVG * ×55) *b*, A large muscular artery It gives rise to a right-angle branch (artero-arteriole) which proximally has the structure of a small muscular artery (see *c*) and distally the structure of an arteriole (see *d*) (hematoxylin and eosin, ×55) *c*, The portions of the large and small muscular arteries shown within the rectangle in *b* The basic structure of the two is similar The presence of muscular media in the branch at this level is in contrast to the absence of an identifiable muscular layer in the same branch more distally as illustrated in *d* (ELVG ×440) *d* The distal portion of the branch arising from the large muscular artery and showing the structure of an arteriole (ELVG ×440)

layer composed of such tissue. It is to be recognized, therefore, that there is in the lung a class of vessel which arises from the large muscular arteries and which proximally has muscle in its wall, while distally it has an arteriolar structure. Although the proximal portion of this vessel may be classified as

* In this paper the abbreviation "ELVG" refers to Verhoeff's elastic tissue stain counterstained with van Gieson's connective tissue stain.

a small muscular artery and the distal portion as an arteriole, it is obvious that one is dealing with different portions of the same vessel. This phenomenon is more readily evident when serial sections of lung, rather than isolated cross sections, are studied This class of vessel might justifiably be designated as an "artero-arteriole " If it is correct to assume that vessels with well defined muscular layers have greater ability to contract than do vessels without such layers, one might transpose the anatomic findings into the functional assumption that a changing tone would be readily possible in the proximal part of the "artero-arteriole" but not in the distal. From the distal portion of the "artero-arteriole" there arise horizontal branches which are the capillaries of the alveolar walls.

The venules originate from confluence of distal portions of capillaries. Histologically the venule cannot readily be distinguished from the arteriolar portion of the artero-arteriole except by the study of serial sections. In chance sections, however, one may suspect the vessel of being a venule by its relatively wide character compared to other vessels of similar structure, and of course it may also be identified as a venule if its connection with a vein is by chance encountered The venules join to form vessels with obvious venous structure.

In a discussion of conditions wherein there is a communication between the two circulations, consideration initially should be given to two factors. (1) the size of the communication[1] and (2) whether or not pulmonary stenosis exists.

THE FACTOR OF SIZE OF COMMUNICATION BETWEEN THE TWO CIRCULATIONS

The cases of "classic" patent ductus arteriosus and the cases ordinarily classified as examples of "uncomplicated ventricular septal defect" usually have essentially normal pulmonary arterial pressures and resistances[2-5] In these conditions the pulmonary vessels are normal histologically[6-7] and the right ventricular thickness is normal In these conditions, from anatomic and functional viewpoints the pulmonary vascular tree does not seem to have any significant function in adjusting to the phenomenon of a communication between the two circulations. The basis for a normal differential in pressure between the greater and lesser circulation seems to be inherent in the resistance imparted to flow through the communication itself (Fig. 2*a*) In the case of ventricular septal defect this is probably related chiefly to the width of the opening and in the case of patent ductus it is related both to the width and length of the ductus The greater the length and the narrower this anomalous channel is, the greater will be the resistance to flow through it. If the resistance to flow through the opening is great enough, a differential in pressure between the two circulations can exist under conditions of the usual ranges of blood flow.

In a rare case of ventricular septal defect or patent ductus arteriosus, pulmonary hypertension is demonstrated or may be assumed when the size of the communication is such that it could have been responsible for a differential in pressure between the two circulations. A case reported by Campbell

and Hudson[8] is one in point, since it involves a patient who at one period had classic signs of an uncomplicated patent ductus arteriosus Later these clinical signs disappeared. At necropsy severe and widespread occlusive lesions were present in the small pulmonary vessels

Such a case is exceptional and in it the pulmonary hypertension may be considered an incidental complication rather than an integral part of the disease. In the usual case of patent ductus arteriosus or ventricular septal defect with pulmonary hypertension, the pulmonary hypertension, on the contrary, is an integral part of the condition More will be said of this type of case in another part of this communication

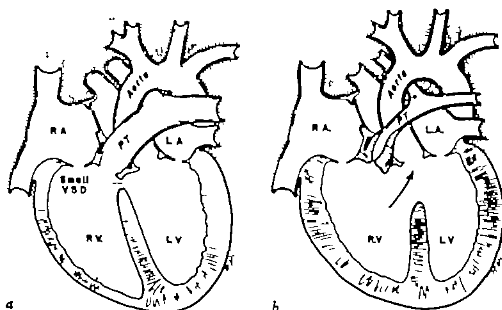


Fig. 2. *a* Ventricular septal defect in which the opening is small. The resistance to flow imparted by the small size of the opening may be considered a factor of adjustment to the presence of the communication. This adjustment allows for a differential in pressure between the two ventricles and may be associated with normal ranges of pulmonary resistance and pressure *b* The tetralogy of Fallot (ventricular septal defect and pulmonary stenosis) The ventricular septal defect is so large that it allows free communication between the two ventricles The pulmonary stenosis is a factor adjusting to this free communication. It allows for right ventricular pressure to equal left ventricular pressure and at the same time for pulmonary resistance and pressure to be normal

THE FACTOR OF PULMONARY STENOSIS

When pulmonary stenosis occurs in association with a communication between the two circulations, the communication is usually in the form of a large ventricular septal defect. Moreover, the combination is generally part of the complex usually designated as the tetralogy of Fallot. The case of infundibular ostial stenosis and ventricular septal defect that Civin and I[9] reported may functionally be considered a variant of the tetralogy In cases of pulmonary stenosis and ventricular septal defect the large ventricular septal defect probably exerts no significant resistance to flow between the two ventricles, so that they are essentially in free communication one with the other

The factor adjusting to the free communication between the ventricles is a level of high resistance to pulmonary blood flow. This high resistance does not occur in the small pulmonary vessels in which normal ranges of resistance, pressure and structural features exist. Instead, the high resistance to flow is in the major pathways to the lung. It resides in the major pulmonary arterial or valvular stenosis or in the subpulmonary stenosis, one of which forms an integral part of the anatomic complex (Fig 2*b*).

It is important to recognize the gross pulmonary stenosis as an effective barrier to pulmonary flow against which right ventricular pressure may be built up to systemic levels. The adjustments are such, therefore, that although a free communication exists between the two ventricles, a left to right shunt

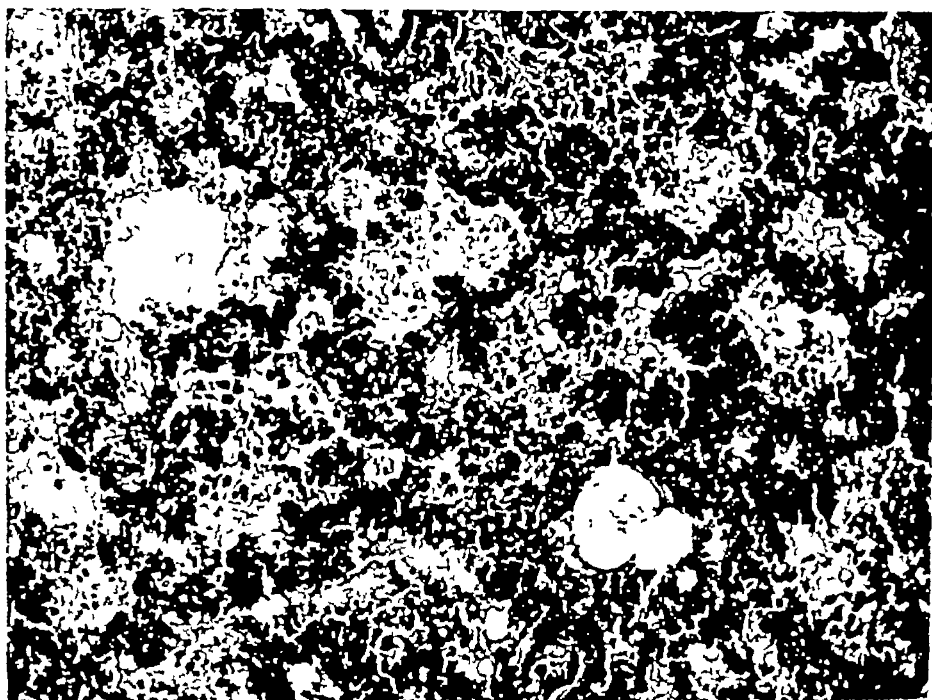


Fig 3 Lung from a 5-month-old infant, with a wide muscular ventricular septal defect and no pulmonary stenosis There are pulmonary congestion and edema, as well as focal hemorrhages (hematoxylin and eosin, ×125)

does not ordinarily occur. In the specimen of the heart in such cases, the right ventricle is about as thick as the left ventricle, just as during life the pressure in these two chambers are about equal.

The concept that the pulmonary stenosis represents an adjustment to the presence of free interventricular communication forms the background for the surgical creation of pulmonary stenosis for large ventricular septal defects.[10]

If congenital pulmonary stenosis accompanying a large ventricular septal defect were effectively corrected and all other conditions remained the same, a tremendous left to right shunt would occur. Such a phenomenon may in fact occur in the situation wherein an infant dies of the effects of a large left to right shunt with pulmonary edema and hemorrhage (Fig. 3) from either a congenital patent ductus arteriosus or a congenital ventricular septal defect without pulmonary stenosis[11-13]

WIDE COMMUNICATION WITH BALANCED CIRCULATION

In striking contrast to the infant with the unbalanced circulation in the situation just mentioned is the patient with a wide communication but with a balanced circulation and with little, if any, left to right shunt. In our necropsy files there are records of 8 such adult patients who were of special interest to Dr Luis M Becu and me from a pathologic standpoint. All of these patients were women. Six had been studied by cardiac catheterization. Since the principal pathologic and clinical features in the other 2 cases were similar to those in the 6 in which physiologic studies were made, these 2 cases will be included in the discussion with the 6

I should like to make particular reference to each of these 8 cases When each of the 6 catheterized patients was studied physiologically by Dr Earl H. Wood, severe pulmonary hypertension was demonstrated. In each patient the pulmonary and right ventricular pressures had been in the same range as the systemic arterial pressures The high pulmonary pressure was associated with *very high pulmonary "arteriolar resistance and not with high pulmonary flow* These features were similar to that in the cases of Eisenmenger complex described by Bing and associates [14] Each patient had had a bidirectional shunt of such a character that the total pulmonary and systemic flows in any case were not greatly different one from the other It is apparent that in these cases the basis for a balanced flow resided in the high resistance to flow, not, however, in the major pathways to the lung as in the tetralogy of Fallot, but rather in small vessels of the lungs (Fig 4)

Four of the catheterized patients had patent ductus arteriosus In each, at the time of operation, the ductus was identified as a short wide vessel the diameter of which was usually similar to that of the aortic arch The ages of those patients were 20, 24, 35 and 45 years respectively *

Of the two noncatheterized patients one was 23 years old and had a patent ductus arteriosus The ductus was of the "window" type, having no measurable length, and its diameter in the pathologic specimen was 0 8 cm.

Of the two remaining catheterized patients one was 33 years old and had a ventricular septal defect which measured 1.5 cm in diameter in the necropsy specimen and lay in the general region of the membranous septum. The other patient, aged 34 years, had a ventricular septal defect measuring 2 5 by 2.0 cm. and a short patent ductus arteriosus Additionally, this patient had evidence of mitral insufficiency resulting from anomalous chordae tendinae of the mitral valve. In this patient the ventricular septal defect lay some distance from the membranous septum. The defect involved the posterior part of the muscular septum and lay inferior to the mitral valve. The remaining noncatheterized patient was a 51-year-old woman who had a patent ductus arteriosus of the 'window" type measuring 1 0 cm. in diameter in the specimen and a muscular ventricular septal defect measuring 1 5 by 1.2 cm.

The 5 specific cases of patent ductus arteriosus (without ventricular septal defect) mentioned above are of the type which in recent years has been variously designated as 'atypical patent ductus arteriosus,' ductus arteriosus

* Two of these cases were reported from the clinical and physiologic viewpoints by Burchell and associates[15] as cases 4 and 7

with reversed flow" and "patent ductus arteriosus with pulmonary hypertension," among other terms. The specific case of ventricular septal defect (without patent ductus) might be called "the Eisenmenger complex" or "ventricular septal defect with pulmonary hypertension."[1,2,16,17]

HISTOLOGIC ASPECTS Of particular interest in the 8 cases just mentioned are the histologic findings in the intrapulmonary arteries. In each case the pulmonary muscular arteries had a thick, muscular media (Fig. 5*a*), this being a feature established from earlier studies as one seen in conditions

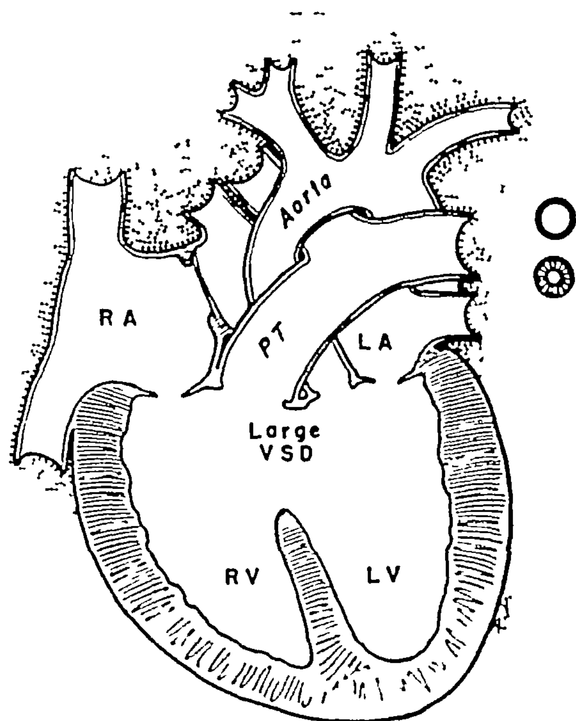


Fig 4. Large ventricular septal defect The large size of the defect allows free communication between the two ventricles There is no pulmonary stenosis The dynamics depend, therefore, upon responses in the pulmonary vascular bed If resistances remain low, a large left to right shunt exists and death may occur in infancy as a result of the effects of the large shunt If pulmonary resistance is at a high level, there may be a balanced type of circulation This phenomenon would be associated with high pulmonary pressure in which the pulmonary arterial pressure is essentially equal to systemic arterial pressure In patients reaching adult life with this phenomenon, the existing medial hypertrophy of the small pulmonary arteries may become complicated by severely obstructing changes involving the intima of these vessels

characterized by a common ventricular ejectile force in the absence of pulmonary stenosis[7,10,18,19] and also being reviewed by Dr. Dammann in another section of this symposium. Similar pulmonary vessels are observed in the crocodile (Fig. 5*b*) and in the turtle (Fig. 5*c*). In the crocodile, as in the alligator, while one aorta arises from the left ventricle, a second aorta arises in company with the pulmonary trunk from the right ventricle. No pulmonary stenosis exists. In the turtle, functional phenomena similar to those of the Eisenmenger complex in man, including the presence of pulmonary hypertension, have been demonstrated.[20] In earlier studies some of

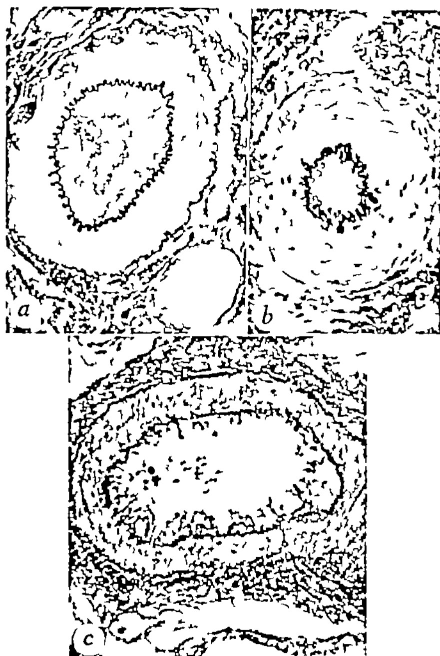


Fig. 5 Muscular pulmonary arteries. *a* From a 35-year-old patient with patent ductus arteriosus and pulmonary hypertension There is pronounced medial hyper trophy of the large muscular artery A small muscular artery beside it shows luminal obstruction by intimal tissue (ELVG ×125) *b* From a specimen of thoracic organs of a crocodile submitted to the author from the Belgian Congo by Drs Marjorie and Howard Horner The thick muscular layer of the media is similar to that seen in the human patient whose lung is illustrated in *a* (ELVG ×245) *c* From an adult American snapping turtle A thick media is present (ELVG ×150)

my colleagues and I demonstrated that among patients having a common ventricular ejectile force the older ones tend to have intimal changes superimposed on medial hypertrophy of the muscular arteries [7 9 18] Such changes were evident in each of the adult cases now being discussed (Fig 5*a*) In the past in our laboratory we have simply called such changes "intimal fibrous proliferation."

The recent studies that Dr Becu and I made of serial sections of the lungs

revealed that changes in the intima of these older patients are more common, and at times more complex, than might be demonstrated by the study of independent cross sections. The obvious reason for this stems from the fact that when arterial lesions occur they are segmental in distribution, and hence observation of a lesion in a chance cross section of a given vessel would depend upon how much of the length of the vessel was involved.

Study of serial sections gave us a greater insight into the varied natures of the obstructive intimal lesions. Additionally, insight was gained into the nature of certain plexiform lesions of the lungs which we and others have observed in cases characterized by severe pulmonary hypertension These lesions have been variously considered to be congenital alterations of pulmonary vessels in the general category of glomus-like bodies, or arteriovenous fistulas,[21] or acquired lesions in the form of recanalized thrombi.[22,23] We believe this lesion to be acquired in nature and have demonstrated that it involves arteries and is common when obstructive intimal lesions exist in patients with severe pulmonary hypertension.

Aside from this peculiar plexiform lesion, which will not be discussed for the moment, we observed that some of the changes in the pulmonary arteries of the 8 adult patients under consideration were similar to changes observed in the systemic circulation in primary malignant hypertension. In this particular group of cases we did not observe acute necrosis of arteries as was reported in single cases of the Eisenmenger complex by Old and Russell[24] and by Kipkie and Johnson.[25] Nevertheless, some of the lesions observed could have resulted from acute arteritis occurring some time earlier.

We have, however, observed acute necrotizing pulmonary arteritis in other adult patients with severe pulmonary hypertension, as in a case of pulmonary hypertension of unknown cause and in a case of atrial septal defect with severe pulmonary hypertension [26]

In 1952 Symmers[27] reported finding in the lungs of 2 patients with pulmonary hypertension necrotizing arteritis similar to that in periarteritis nodosa, and also changes similar to those in the systemic circulation in systemic hypertension. One of his patients was a man, 24 years of age, with pulmonary hypertension of undetermined cause, and one was a man, 34 years of age, with mitral stenosis. Hicks reported a similar case.[28]

In our cases the changes comparable to those seen in systemic hypertension included nonspecific intimal fibrous thickening and hyalinization of the preexisting elements of the vascular wall associated with intimal thickening by amorphous hyalinized material (Fig 6*a*). Arterial aneurysms, scarring (Fig. 6*b*) and thrombosis (Fig. 6*c*) and the plexiform lesions to be described in greater detail may have resulted from earlier acute arteritis.

Plexiform Lesions. The lesion which was common and of considerable interest was the plexiform one in which there were vascular spaces with walls usually composed of varying-sized sheets of endothelial cells. These lesions were found in large numbers in each of the 8 adult cases discussed here and represented the major type of lesion causing organic obstruction of the small pulmonary vessels These are the lesions, as I have indicated, which have been called at times arteriovenous connections and at times glomus tumors

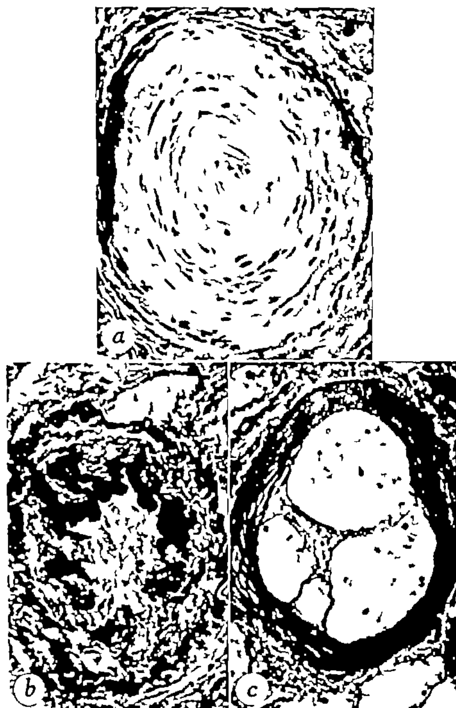


Fig. 6 *a*, A small muscular artery from a 24-year-old patient with patent ductus arteriosus and pulmonary hypertension There is hyalinization of the arterial wall with luminal occlusion (hematoxylin and eosin ×260) *b* From the 24-year-old patient with patent ductus arteriosus A large muscular artery shows intimal fibrous thicken ing and medial scarring, as well as medial hypertrophy (ELVG, ×260) *c* A small muscular artery from a 45-year-old patient with patent ductus arteriosus Recanaliza tion of a thrombus is shown (ELVG· ×200)

or glomus-like lesions of the lungs in patients with severe pulmonary hypertension (Fig. 7) From examination of individual cross sections it is impossible to arrive at any concrete conclusion regarding the nature and location of these lesions, but with serial sections several facts about them were readily brought out. Immediately apparent is the fact that these plexiform lesions were con-

fined to arteries, and more specifically to the proximal portions of the artero-arterioles (Fig. 8), a feature also observed previously by others[22,29,30] We were unable to demonstrate any connection between these lesions and veins

The question arose whether the plexiform lesions represented organized thrombi, as suggested by others[16,22] Our feeling was that they did not represent organized thrombi, but rather that their evolution involved necrosis or hyaline alteration in the arterial wall, followed by endothelial proliferation of a villous character. The evidence suggested that hyalinized or fibrinoid material in the vascular wall was irritative to the surrounding endothelium, causing

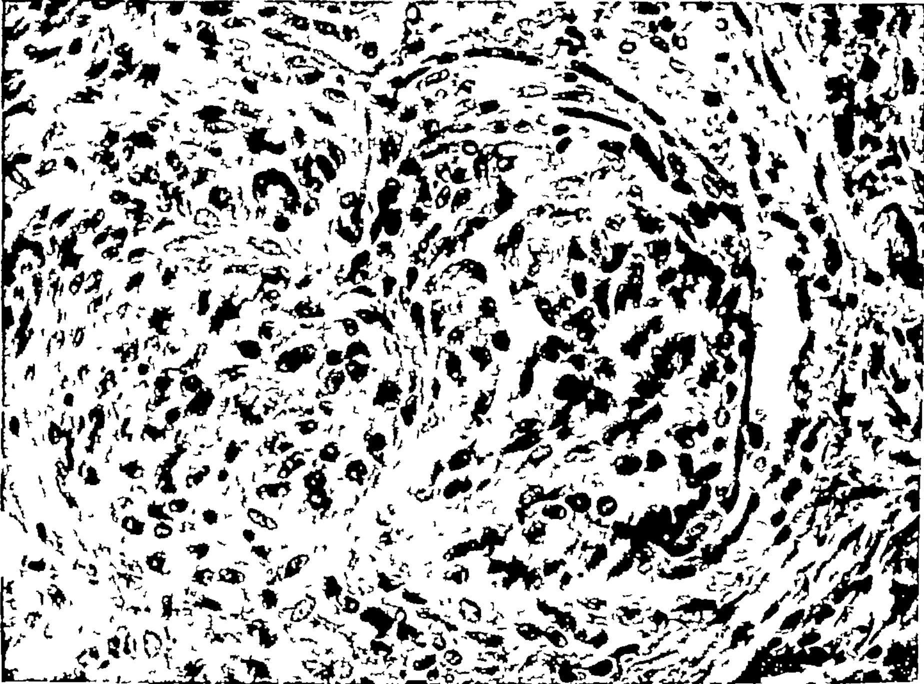


Fig 7 A plexiform lesion of the lung involving a small muscular artery This peculiar lesion is made up of sheets of cells, between which are blood spaces In this section no definite arterial wall is identifiable The location and nature of such a lesion can be ascertained by a study of serial sections as illustrated in Fig 8 This section is from a 23-year-old patient with patent ductus arteriosus and clinical and pathologic evidence of pulmonary hypertension (hematoxylin and eosin, ×320)

it to proliferate in this peculiar manner Frequently there were areas of loss of arterial wall in relation to these lesions In some instances hyalinized material was related to the proliferated cells.

Comparison Between Adults and Children. We compared our findings in these 8 adult patients who had severe pulmonary hypertension with patent ductus arteriosus or ventricular septal defect or both, as one group, with infants and children having anatomically comparable defects, as another group Included in the latter group were 4 patients who by cardiac catheterization studies were shown to have pulmonary hypertension and left-to-right shunts. The anatomic malformations and the ages of the latter patients were as follows patent ductus arteriosus in two 3-month-old infants, ventricular septal defect in one 3-month-old infant, and endocardial sclerosis and coarctation of the aorta opposite a patent ductus arteriosus in a 5-year-old boy In each there was medial hypertrophy of the muscular arteries, but there

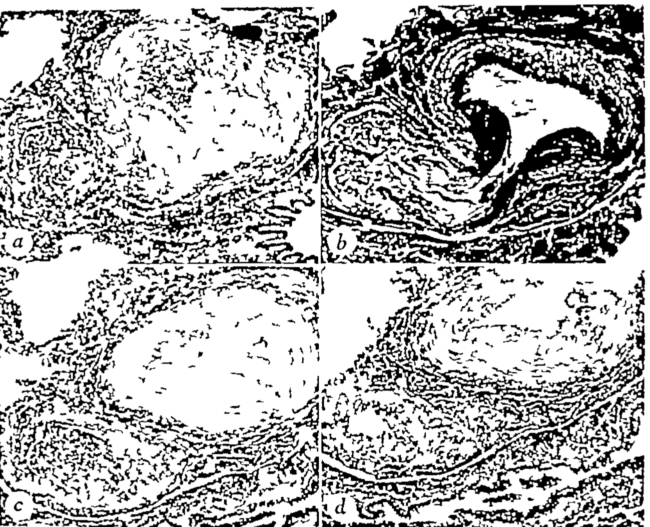


Fig 8 Muscular arteries of the lung from a series of sections from the same patient as in Fig 7 *a* Beside a large muscular artery with medial hypertrophy there is a plexiform lesion like that illustrated in Fig. 7 *b*, This lesion is shown to involve a small muscular artery as it arises from the large muscular artery At this level the intima of the large muscular artery shows hyalinization around the ostium of the branch. *c*, This section lies at a level beyond that in *b* The plexiform lesion is shown at a level where part of the wall of the artery is identifiable, while elsewhere the lesion is not related to identifiable arterial wall. *d* At a more distant level the arterial lumen is wider and the structure of the wall approaches normal. The series illustrates the arterial origin of the plexiform lesion and it also represents the segmental nature of the obstructive lesion. (All sections are magnified 90 times; *a*, *c* and *d* are stained with hematoxylin and eosin and *b* is stained with ELVG)

were none of the intimal and obstructive luminal changes observed in the adult patients.

These observations lead to the conclusion that the various lesions associated with intimal thickening and luminal obstruction are neither congenital nor an integral part of the condition. Rather, these changes seem to be acquired complications of the existing pulmonary hypertension and seem to cause a fixed reduction in the size of the pulmonary vascular bed. The medial hypertrophy of the muscular arteries, on the contrary, is a universal finding in patients having a free communication and pulmonary hypertension is an integral part of the disease complex. The intimal changes may perhaps underlie the increasing degrees of right to left shunt that may characterize the course of these patients with the passage of time, and also these intimal changes may underlie the phenomenon, now generally recognized, of the poor



Fig 9. The thoracic organs viewed from in front in the case of a 52-year-old woman with ventricular septal defect, patent ductus arteriosus and anatomic and clinical evidence of pulmonary hypertension Wide pulmonary trunk

outlook for the adult patient with patent ductus arteriosus, pulmonary hypertension and a right to left shunt in whom the ductus is closed surgically.

The correlation may be carried further to the generally good prognosis that is associated with surgical closure of a patent ductus in a child or infant even when pulmonary hypertension is shown to be present. The era of closure of ventricular septal defects is upon us; the foregoing remarks regarding patent ductus arteriosus seem also to apply to ventricular septal defect.

GROSS PATHOLOGIC FEATURES. The gross pathologic features of the cases in which there are free communication between the two circuits and pulmonary hypertension are also of interest. As is apparent in the roentgeno-

grams of such patients, the pulmonary trunk and the major pulmonary arteries are greatly dilated (Fig 9) My colleagues and I have not ourselves made organized investigations of the gross appearance of the small pulmonary vessels in these conditions, but insight into them may be gained from the



Fig. 10 From a 35-year-old catheterized patient with patent ductus arteriosus and pulmonary hypertension. *a* The tracheobronchial tree and major pulmonary arteries viewed from in front. The pulmonary trunk had been divided just above the valve and has been reflected upward as has been the left pulmonary artery This allows the concavity of the left bronchus caused by the left pulmonary artery to be seen. The intimate relations between the right pulmonary arteries and the branches of the right bronchus are also evident. *b* Roentgenogram of the specimen of trachea and major bronchi showing the indentation along the left side of the trachea which had been caused by the aorta. Also shown is the concave deformity of the left bronchus caused by the left pulmonary artery

report of Hultgren and associates[31] and of Yu and associates [32] Each report refers to an adult case in which there were patent ductus arteriosus and pulmonary hypertension and in which, with the necropsy specimen, plastic material was injected into the pulmonary arterial system followed by digestion of the lung. In each of these there was a paucity of the fine branches,

many of which characterized the pattern of the normal. These findings probably are to be correlated with the observed tendency for the seriously obstructing arterial lesions occurring in such cases to exist at the origins of the branches arising from the large muscular arteries.

The dilated major pulmonary arteries have evident effects upon the tracheobronchial tree with which they are in direct or indirect contact. The major bronchi and their branches may show alterations in their contour and caliber resulting from the pressure exerted by the increased size of the large pulmonary arteries (Fig 10*a*). Focal narrowing of the bronchial tree may perhaps be the cause of localized emphysema which is sometimes seen in such patients.

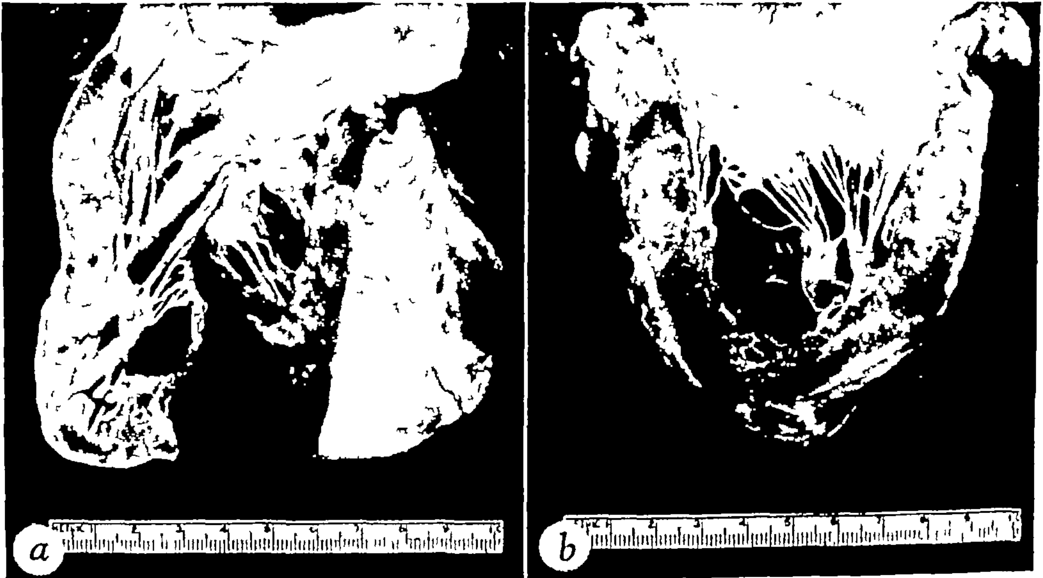


Fig 11 From a 20-year-old catheterized patient with patent ductus arteriosus and pulmonary hypertension *a*, Right ventricle There is marked concentric hypertrophy in which the thickness of the wall approximates that of the left ventricular wall illustrated in *b* With regard to thickness, the relationships between the two ventricles in this adult patient are similar to those of the normal fetus This relationship is also consistent with the fact that during adult life the pressures exerted by the two ventricles were essentially equal

Special mention should be made of the effects upon the trachea itself (Fig 10*b*) In cases of the type being discussed, the left side of the trachea frequently shows an indentation at the level of the aortic arch This appears to result from upward and medial deviation of the aorta as a result of pressure against its lower left aspect by the enlarged pulmonary trunk and left pulmonary artery It is pertinent to recall that at the level where the aortic indentation is apparent in the left side of the trachea, the left recurrent laryngeal nerve runs between the aorta and trachea In the cases of pulmonary hypertension in which there is paralysis of the left vocal cord, the basis for the abnormality may perhaps reside in pressure upon this nerve as it lies in this position

The appearance of the heart either in ventricular septal defect with pulmonary hypertension or in patent ductus with pulmonary hypertension differs

from the appearance in either of these conditions without pulmonary hypertension. In the presence of pulmonary hypertension there is striking concentric hypertrophy of the right ventricular wall, the thickness of this chamber being essentially equal to that of the normally thick left ventricle (Fig 11) Consistent with the fact that usually in these cases the volume of flow through each side of the heart is about equal and near normal is the fact that the cardiac chambers are within normal limits as to size.

SUMMARY

When a communication exists between the two circulations, either at the ventricular level or between the aorta and the lesser circulation, adjustment to the communication may reside in the opening itself, in associated pulmonary stenosis or in the small pulmonary vessels When the opening between the two circulations is sufficiently narrow, the opening itself displays sufficient resistance to flow for the pressure and resistance in each circulation to be essentially normal

If a large opening exists between the two ventricles and there is also pulmonary stenosis, the latter may be considered as an adjusting factor to the communication between the two ventricles The pulmonary stenosis allows for a normal pulmonary pressure, while the pressures in the two ventricles are about equal

When there is free communication between the two circuits and no pulmonary stenosis, adjustment depends upon responses in the small pulmonary vessels When an adjustment occurs to allow a balanced flow, the anatomic picture in the small vessels is that of medial hypertrophy of the muscular arteries Such an adjustment being manifested by high resistance to pulmonary flow is associated with pulmonary hypertension, the pulmonary pressure being in essentially the same range as the systemic arterial pressure. Such a functional arrangement leads eventually to the development of secondary lesions in the pulmonary vascular tree. These consist of a variety of lesions, all of which are influential in causing organic obstruction to the flow within the pulmonary vascular bed

The key to prognosis in patients having free communication between the two circuits and a balanced flow seems to reside in whether or not the pulmonary hypertension has caused sufficient secondary occlusive changes to bring about permanent and significant reduction in the size of the pulmonary arterial bed. In general, the intimal changes are seen only in the older patients among those having balanced flows and pulmonary hypertension.

Gross distortion in the tracheobronchial tree occurs as a result of enlargement of the pulmonary arteries

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Question (Chairman Bing) From the discussion by Dr Dammann and Dr Edwards one gets the very definite impression that increase in flow through the lung or pulmonary blood flow is responsible for these changes that you have observed—the lumen wall ratio Does that agree with the concept of Dr Edwards? Dr Dammann, will you comment on that, please?

Answer (Dr Dammann) I don't agree with that thought. I think primarily it is an increase in pressure If you have a single ventricle or a large ventricular defect, no matter what the resistance is at the end of the line, the pressure has to be the same on both sides It has to be the same in the pulmonary artery and aorta.

The flow may differ The flow will depend upon resistance. I think it is the prolonged pressure and high pressure that effects change. I do not know of any system in the body where we have proof that flow alone causes change. In each instance it is pressure

Answer (Dr Edwards) The question is rather complicated, but perhaps we can simplify the answer a bit

First of all, when there is a free communication between the two circulations, either a ventricular level or ductus, all patients have medial hypertrophy of the muscular arteries of the lung I think Dr Dammann has had such experience and will agree. We have suggested in the past that this change was similar to the change which occurs normally in the pulmonary bed in the fetus The fetus, of course, has pulmonary hyper-

tension in that its pulmonary pressure is the same as the systemic pressure, and there is free communication at ductal level

This medial hypertrophy that occurs in the pulmonary vascular bed can be compared to the right ventricular hypertrophy which is normal in the fetus and in these cases that have a free communication with elevated pressure in the postnatal life

I think perhaps it is well to consider pulmonary arteries as a prolongation of the right ventricle They are almost part of the right ventricle. When there is thickening of the right ventricle from whatever cause, the pulmonary arteries in the medial layer are thick It is hard to say which comes first, but I think it is easier to look upon it as an integral part of this malformation

I don't think we want to throw the lung away and just look at the heart We have to look at both as one complex Medial hypertrophy, then, is part of the complex

With it, physiologically there is an elevation of pulmonary arterial pressure. From the observations that we have made it appears that if pulmonary hypertension is present long enough, and if the pulmonary hypertension is high enough, then we may get these secondary changes. These resemble some of the changes we see in systemic circulation, in systemic hypertension Some of them resemble the changes seen in periarteritis nodosa

These changes are not restricted to patients who have congenital malformations and pulmonary hypertension, but they are also seen in other conditions when there is a markedly elevated pulmonary arterial pressure for a long period of time. They are seen at times in mitral stenosis (not very commonly, but they are seen), and they are seen also in patients who have so-called idiopathic pulmonary hypertension

So, I think we have to attribute these secondary occlusive changes in the pulmonary bed not to the increased flow, if it ever occurs (and probably it does in some of the patients for some periods of time), but rather to the existing elevation in pulmonary arterial pressure So, in that regard as in many others, I would agree with Dr Dammann

I would like to mention one other thing In our recent studies on the pulmonary bed, including some of the serial section work, I have had very happy collaboration on it by Dr Luis Becu Castro, who is working with us this year, and who comes from the Argentine

LEFT TO RIGHT SHUNTS IN INFANCY

HELEN B TAUSSIG (*Baltimore*)

After the first two papers, I am very humble in trying to discuss with you left to right shunts in infancy in 15 minutes Such shunts in infancy may be very hard to differentiate one from another clinically However, let us review briefly the main groups

As Dr Dammann has already mentioned, a large patent ductus arteriosus may produce a large left to right shunt in infancy I am speaking now of large shunts because I think the smaller ones, when a patient is doing well, can await the growth and development of the child to clarify the clinical picture and do not need to cause us great concern at this point. In addition to the large patent ductus, perhaps the next most common problem is that of the large interventricular septal defect with increased pulmonary blood flow and high pulmonary pressure, including the so-called Eisenmenger group Then there is the single ventricle and finally septal defects above the atrioventricular valves, that is, atrial septal defects and anomalies of the pulmonary venous return.

LARGE PATENT DUCTUS ARTERIOSUS In the noncyanotic child, who has evidence of cardiac enlargement and increased pulmonary blood flow, the large patent ductus is usually considered to show electrocardiographically a balanced axis in the extremity leads and combined ventricular hypertrophy in the precordial leads A large ventricular septal defect may give very much the same picture. I do not think we can always tell them apart clinically Both of these conditions have a systolic murmur and accentuated pulmonic second sound and frequently an apical mid-diastolic murmur Therefore clinically and auscultatorily they are similar and require special studies for differentiation.

Cardiac catheterization will help in that in ventricular septal defect one expects a greater oxygen increase in the ventricle than in the pulmonary artery, whereas the reverse is true in patent ductus arteriosus This by no means clearly separates the two groups, however, because occasionally in the high ventricular septal defect the major oxygen step-up may be in the pulmonary artery On the other hand, in a ductus with pulmonary regurgitation, the oxygen step-up may be in the ventricle.

We have, therefore, followed Dr Keith's principle of doing retrograde aortograms with 35 per cent Diodrast to determine whether there is simultaneous visualization of the aorta and pulmonary arteries, in other words, dye filling the lung vasculature via the patent ductus The difficulty here is that such simultaneous visualization may not differentiate between a ductus and

an aortic septal defect, a problem which may be solved by improved angiocardiographic technique. Another important thing to remember is that after dye has been shown to pass from the aorta to the lungs, as in a patent ductus, the possibility of an additional malformation below the aortic and pulmonary valves, such as a *high interventricular septal defect* or even a single ventricle, has not been ruled out It is obviously foolish to attempt surgery on a patent ductus when there is so serious a residual defect as a single ventricle remaining

The problem therefore is not easy, and yet fortunately, if there is evidence of a large ductus, the chances are all in favor of greatly helping the child by closure of the ductus In most instances we are gratified to find that there are no associated major defects in the ventricular septum and the patient is therefore greatly helped

VENTRICULAR SEPTAL DEFECTS. With ventricular septal defects, particularly with high pulmonary pressure and vascular lung fields, it is more common to see abnormal degrees of axis deviation on the electrocardiogram with widening and significant notching of the QRS complex than in the simple patent ductus The aortogram, of course, will not show the dye simultaneously opacifying the aorta and pulmonary artery Furthermore, cardiac catheterization will usually help. Usually in this group systemic arterial oxygen saturation is normal. Early visualization of the aorta does not occur with venous angiocardiography. In a later symposium we will hear more about the possible surgical correction of ventricular septal defects.

Another important question is that of surgically decreasing pulmonary pressure to protect the lung circulation, and I hope Dr. Dammann and Dr Morrow will have something more to say about this work.

SINGLE VENTRICLE The above malformation must be differentiated from the single ventricle without clinical signs and with increased pulmonary blood flow in which one may have simultaneous visualization of the aorta and pulmonary artery angiocardiographically In this group, one's only hope is to break the pulmonary artery pressure and decrease the circulation to the lungs, thereby diverting more blood out into the aorta

ATRIAL SEPTAL DEFECTS. We come now to the group of atrial septal defects which, in infancy, may or may not be associated with a murmur. I think it is seldom that one finds a large ductus causing clinical difficulty and with no murmur whatever Also, one seldom finds a large ventricular defect with no murmur However, a large atrial septal defect may occur without a significant murmur. Usually the electrocardiogram shows right axis deviation in the extremity leads and right bundle branch block in the precordial leads, without the picture of marked right ventricular hypertrophy This is a situation in which the right ventricle is circulating a large volume of blood with normal or only slightly increased pressure.

We have found, as have others, that children with the so-called ostium primum defect, as opposed to the usual atrial septal defect, have a greater degree of left axis deviation in the extremity leads, signs of right ventricular hypertrophy in the precordial leads and a murmur which is more like that of a ventricular septal defect

ANOMALIES OF PULMONARY VENOUS RETURN. The other condition that

gives great trouble in infancy is anomalous pulmonary venous return. These anomalies are many and varied, ranging from that involving a single lobe to anomalous drainage of the entire pulmonary venous return. We are trying to learn to recognize such anomalies because of their surgical importance. Dr Bahnson and others have made great strides in operating on such defects Incidentally, one of the most serious questions in this regard is how well developed the left side of the heart is and how readily mixed arterial and venous blood can come back to the left side of the heart. In early infancy, those most likely to be in trouble probably have very small left atria and ventricles, which makes operation a great deal more difficult because survival depends mainly on the right side of the heart and perhaps the presence of a large ductus with circulation out through the ductus to the systemic circula-



Fig 1 Roentgenogram of a child with interatrial defect

tion. In this malformation we have had great help from angiocardiography, either through failure to visualize a well developed left atrium or visualizing dilution of the blood in the right atrium at the site of the entrance of the anomalous veins

I should like to present a few instructive cases

Figure 1 shows the x ray photograph of a child with a large interatrial septal defect, but the picture is nonspecific, with a full cardiac silhouette, increased prominence of the pulmonary conus and increased vascularity of the lung fields This is representative of the group in which special studies are usually necessary Figure 2 is typical of anomalies of the pulmonary venous return. In our homely way, we call it a snow ball man with his head off center One circle is above the other circle, eccentrically placed. This is quite characteristic when all of the pulmonary veins drain into the superior vena



Fig 2 Roentgenogram of child with anomalous pulmonary venous drainage (age 4 years).



Fig 3 Enormous dilatation of the right atrium in an infant of 2 months with anomalous pulmonary venous drainage

cava. In early infancy one may not see the ' snow ball", it is only later when the diaphragm begins to drop that this is likely to be seen. Figure 3 also is the x-ray picture of an anomaly of the venous return with great dilatation of the right atrium and rather wide base with perhaps both the right and left superior venae cavae, into which the pulmonary veins drain.

Finally, having tried to define the groups with increased pulmonary blood flow, I would like to show a few complicated situations I am glad to be able to show these in 1955 rather than 1945, since we can cope with them better now



Fig 4 Chest film of infant P H at age 5 months.

An infant (P H) was first seen at the age of 5 months, at which time the roentgenogram showed a large heart and apparent increase in pulmonary vascularity (Fig 4) There was no cyanosis and the electrocardiogram showed right axis deviation. Eleven months later, the heart was even larger and the lung fields still showed signs of vascularity (Fig 5) There was no cyanosis The child was anemic, and with correction of this, his health improved. Eight months later, when the child was 2 years of age, the situation was quite different. The roentgenogram showed a small heart and clear lung fields (Fig. 6) There were definite cyanosis and attacks of paroxysmal dyspnea. Angiocardiography was carried out, and this showed simultaneous visualization of the aorta and pulmonary artery Cardiac catheterization showed a drop in pressure from the right ventricle to the pulmonary artery Thus, the child fulfilled all of our criteria for the diagnosis of the tetralogy of Fallot. The most



Fig 5 Same child as in Fig 4, at age of 16 months

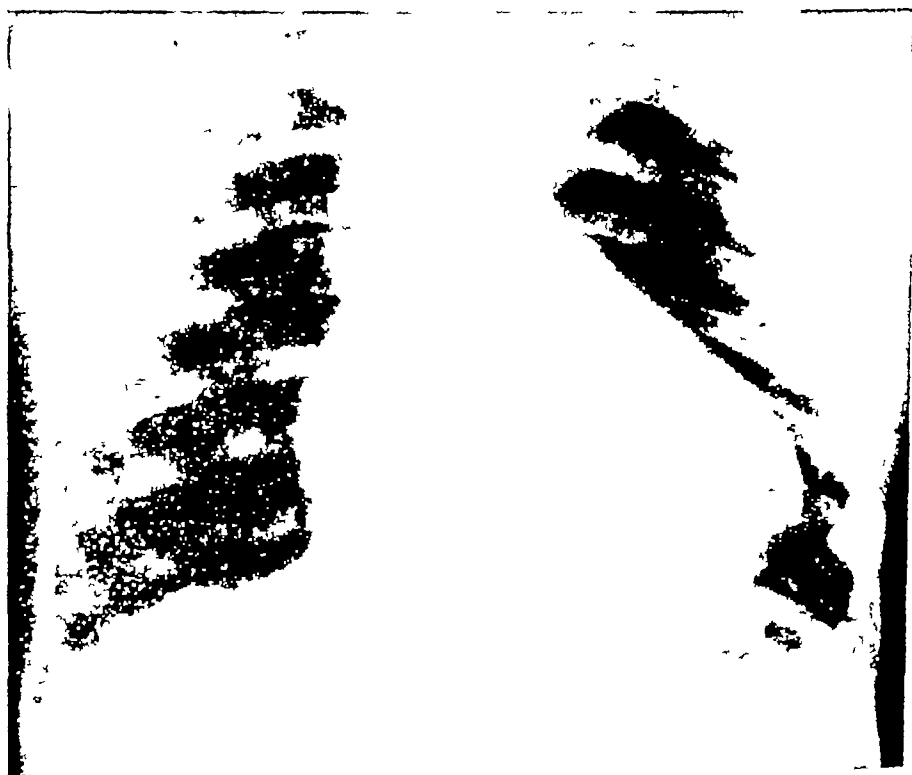


Fig 6 Same child, at age of 2 years Final diagnosis, tetralogy of Fallot

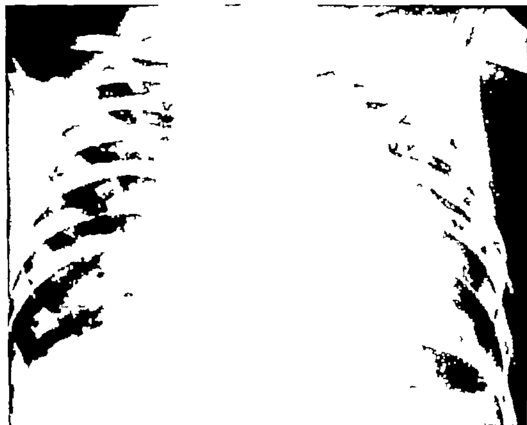


Fig 7 Roentgenogram of chest of infant L J at age of 6 months.



Fig 8 Same child as in Fig. 7 at age of 15 months



Fig 9 Retrograde aortogram (L. J , at age of 15 months), showing that no ductus arteriosus is present

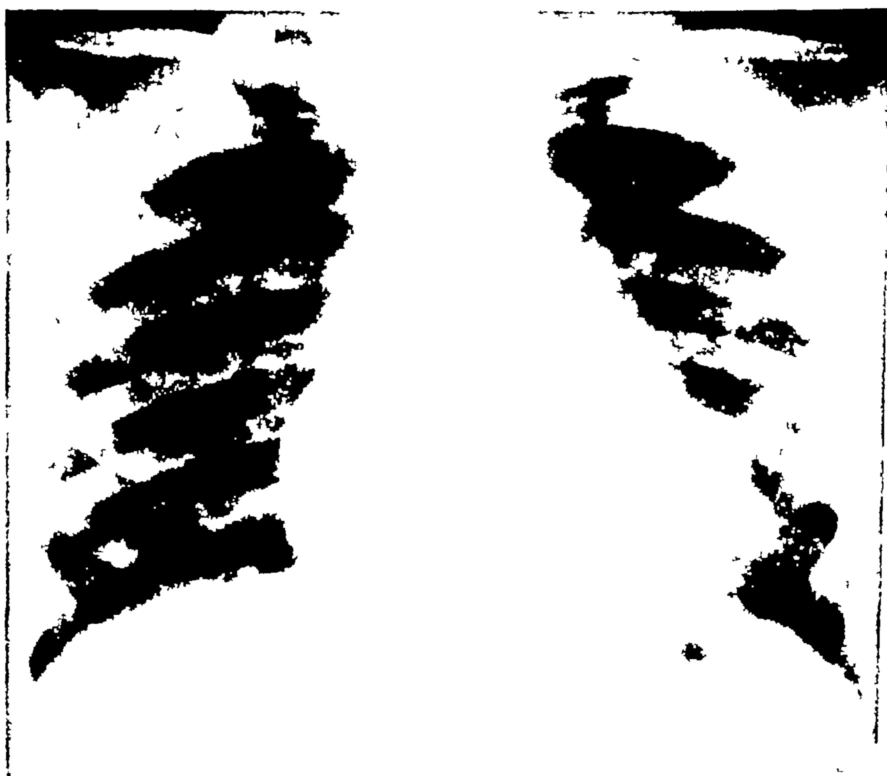


Fig. 10 Same child (L J.) at age of 2 years. The clinical diagnosis of tetralogy of Fallot was made at this time.

difficult thing to explain was not the initial absence of cyanosis but the initial presence of the increased vascularity of the lungs

Another infant (L J) was seen at the age of 6 months The roentgenogram showed a large heart, prominent pulmonary conus and vascularity of the lung fields (Fig 7) There was a gallop rhythm and a systolic murmur, with slight right axis deviation in the electrocardiogram, more than one usually sees in a patent ductus arteriosus Nine months later, the film shown in Fig 8 was made. Again we noted the full heart, prominent pulmonary conus and vascular lung fields There seemed to be slight left atrial enlargement Because some cases of patent ductus may have right axis deviation in the electrocardiogram, a retrograde aortogram was done (Fig 9) This showed that no ductus arteriosus was present, and we made a tentative diagnosis of interventricular septal defect However, when we saw the child at the age of 2 years, the picture had changed considerably There was a small heart (Fig 10), clear lung fields, fairly marked cyanosis and an erythrocyte count of 8 million. She squatted when she was tired and cardiac catheterization showed definite evidence of pulmonic stenosis Evidently, we had been dealing with a case of tetralogy of Fallot!

These two cases illustrate the difficulties of diagnosis in the noncyanotic child with increased vascularity of the lung fields

DISCUSSION

Rodolfo Kreutzer (*Buenos Aires*)

I will limit myself to the consideration of the persistent ductus arteriosus in infants, and especially to the large ductus with early signs of heart failure, which we call "malignant ductus "

In 4 infants in which this cardiopathy was undiagnosed and in which the patent ductus arteriosus was the sole cause of death, the autopsy showed evidence of hypofunction of the foramen ovale during fetal life. We think that this hypofunction, or premature closure of the foramen ovale, is one of the factors which could explain the presence of these enormous ductuses, sometimes of a caliber equal to that of the aorta. Thus, during fetal life, a greater proportion than normal of the blood flows through the pulmonary artery, dilates the ductus and increases the pulmonary circulation This increased pulmonary circulation could prevent the thickening of the media typical in the pulmonary arterioles of the normal newborn. After birth, this dilatation of the pulmonary arteriolar tree permits a large left to right shunt through the large ductus This shunt is the cause of the early left ventricular failure.

Concerning the diagnosis, I would like to point out some of the findings made in the analysis of 30 patients with patent ductus arteriosus seen for the first time when less than one year of age:

1 Only 16 had a continuous murmur
2. Seven had occasional cyanosis

3. In 26 the electrocardiogram showed a large R and a large S in V_1 and small Q, large R and small S with positive T in V_6
4. Cardiac failure was observed in 11 patients, mostly of the paroxysmal dyspnea type, and occasionally of the congestive type.
5. All the patients had cardiac enlargement and increased pulmonary circulation
6. Twenty were females.
7. In 8 there was a history of German measles in the mother during pregnancy.

John Keith (*Toronto*)

We have had 4 cases of tetralogy with increased pulmonary flow. As Dr Taussig has indicated, they are similar to ventricular septal defect

A couple of points that are useful in diagnosis are, first, that half of our cases have a right aortic arch. That is very suggestive of a tetralogy, even though you think it is a ventricular septal defect Second, they have more right ventricular hypertrophy

"ATYPICAL" PATENT DUCTUS ARTERIOSUS

ROBERT F ZIEGLER (*Detroit*)

One cannot define an "atypical patent ductus except by comparing or contrasting it with a "typical' one By the latter terminology is usually understood the clinical syndrome as it is most frequently observed in older children and adults, consisting of absence of cyanosis, the presence of a continuous systolic and diastolic murmur at the upper left sternal border, wide systemic arterial pulse pressure, increased pulmonary vascularity, and enlargement of the left atrium and ventricle Physiologically this "typical" situation exists when pressure-flow relationships in the systemic and pulmonary circulations are also typical, pressure in the former exceeding that in the latter throughout practically the entire cardiac cycle, *and at normal levels*

The first and, in a sense, perhaps the most atypical situation is that in which a continuous aortic-pulmonary artery pressure gradient is maintained at an abnormal level via a complicated patent ductus arteriosus In terms of pulmonary artery pressure there are, of course, two possibilities (1) unusually low pressure as in pulmonary stenosis, particularly the tetralogy of Fallot, and (2) pulmonary arterial hypertension combined with greater aortic hypertension, as in coarctation of the aorta In both cases there will be a continuous murmur and little, if any, cyanosis at rest. In both, in contrast to the "typical" situation, there will be associated, if not predominant, right ventricular hypertrophy as evidenced in the precordial lead electrocardiogram. In each of these syndromes one can easily recognize the presence of a patent ductus, yet there are sufficient clinical and physiologic differences to make it equally apparent that neither of these conditions is typical of an uncomplicated ductus and that they may well require quite different surgical management.

Thus far we have considered briefly two representative syndromes which bear at least a superficial clinical resemblance to an uncomplicated patent ductus arteriosus, but which are significantly different anatomically, physiologically and with respect to surgical management Now, what about those situations in which there is just an uncomplicated patent ductus anatomically but in which the clinical picture differs from that previously described as "typical"? Here again we must concern ourselves with variations in the pressure-flow relations between the systemic and pulmonary circulations, and more particularly the causes and effects of various degrees of pulmonary arterial hypertension. Two syndromes, each different from the so-called "typical patent ductus arteriosus previously described but each absolutely typical and recognizable for what it represents, here require description.

PATENT DUCTUS ARTERIOSUS WITH MODERATE OR MARKED PULMONARY ARTERIAL HYPERTENSION AND INCREASED PULMONARY BLOOD FLOW This syndrome is most typical of uncomplicated patent ductus arteriosus in infants and young children but may also be observed in older children and adults It is characterized physiologically by a large volume of total pulmonary blood flow with an aortic-pulmonary shunt occurring primarily in systole because of the predominantly diastolic pulmonary hypertension. The typical ductus murmur in this situation is therefore systolic rather than continuous Since



Fig 1 Postero-anterior chest roentgenogram in a 36-year-old white male patient with a window type of patent ductus and marked pulmonary artery hypertension with increased volume of pulmonary blood flow The x-ray shows marked cardiac enlargement involving primarily the left ventricle and tremendous increase in pulmonary vascular markings This represents a reversible situation with surgical interference urgently indicated

there is increased volume of pulmonary blood flow and increased return flow to the left atrium and left ventricle as in the syndrome of patent ductus arteriosus with a normal or only slightly elevated pulmonary artery pressure, there will be increased pulmonary vascularity roentgenographically, and enlargement of the left atrium and ventricle by roentgenography or more definitively by precordial lead electrocardiography (Fig 1) Because of the pulmonary hypertension there may also be electrocardiographic evidence of associated but not predominant right ventricular hypertrophy (Fig 2). The pulmonary artery hypertension in this syndrome results primarily from increased flow with normal or only slightly increased arteriolar resistance or from left-sided

cardiac decompensation This is therefore a capillary or post-capillary hypertension, evidence for which is an elevated pulmonary venous pressure as measured by catheterization of the left atrium through a patent foramen ovale or by recording the pulmonary artery wedge pressure An important concomitant auscultatory sign in this situation is the frequently heard mid-diastolic apical murmur of so-called relative mitral stenosis

The therapeutic implications of this type of patent ductus are worthy of special emphasis In the first place, in view of the particular physiopathology which is basically reversible, namely increased volume flow through the ductus and pulmonary circulation, surgical correction is more urgently indicated than in the type of patent ductus with relatively normal pulmonary

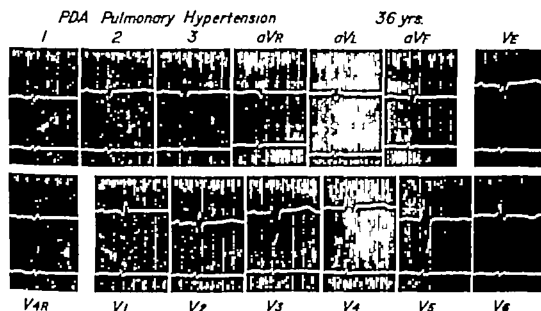


Fig. 2. The electrocardiogram in the same patient as in Fig 1 There is evidence in the unipolar precordial leads of combined right and left ventricular enlargement with late activation time in leads from both right and left sides of the precordium inverted T waves in left precordial leads, and an initial Q in right precordial leads

pressures Secondly, in this type the surgeon may expect to encounter a large short ductus or one of the so-called window or fistulous varieties requiring very careful dissection and division instead of suture-ligation

Patent Ductus Arteriosus with Marked Pulmonary Arterial Hypertension and Normal or Decreased Pulmonary Blood Flow This syndrome is characterized physiologically by an increased pulmonary arteriolar resistance with normal or decreased volume of pulmonary blood flow and equalization or reversal of the usual pressure gradient between aorta and pulmonary artery It is important to note that the level of pulmonary artery pressure does not distinguish this syndrome from the preceding one with pulmonary hypertension and increased volume of pulmonary blood flow The principal differential feature is a normal or decreased pulmonary wedge pressure (or pulmonary venous pressure if available) indicative of a precapillary pulmonary hypertension, in contradistinction to the capillary or post-capillary

hypertension of the previously described syndrome. Other character features related to the increased pulmonary arteriolar resistance include creased pulmonary vascularity roentgenologically (Fig 3), and predomin right rather than combined right and left ventricular hypertrophy elec cardiographically (Fig 4) Other associated but relatively less important s are (1) Murmurs which may be absent altogether, variable systolic, or diastolic murmur of relative pulmonary insufficiency, (2) normal or poss decreased systemic arterial pulse pressure, (3) differential cyanosis of

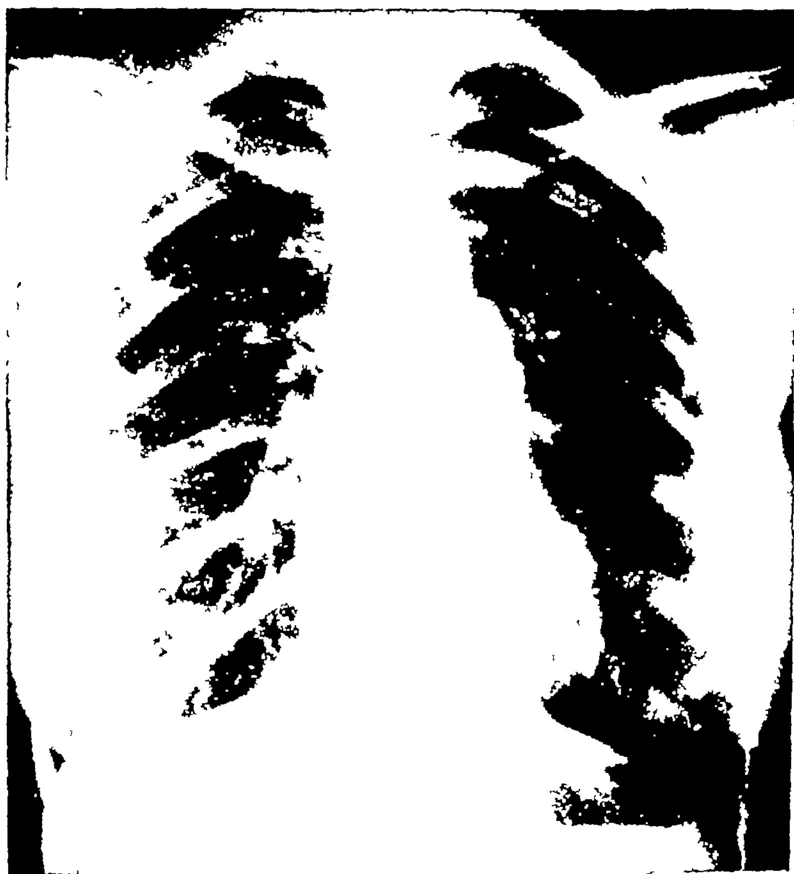


Fig 3 Postero-anterior chest roentgenogram in a 32-year-old white female pati with a patent ductus with marked pulmonary artery hypertension and decrea volume of pulmonary blood flow Gross heart size is not enlarged and there is pro nence of the central pulmonary arteries but a marked decrease in peripheral p monary vascularity—a typical roentgenographic finding in precapillary pulmon hypertension or primary pulmonary endarteritis

lower extremities due to the shunting of unoxygenated blood from the pu monary artery into the descending aorta at rest or induced by exercise, (possible cyanosis (or arterial oxygen unsaturation) in the upper extremiti due to inadequate pulmonary blood oxygenation

Regardless of the pathogenesis of this typical syndrome, that is, whether represents a primary pulmonary endarteritis with a coincidental patent ducti or whether it is the end stage of progressive pulmonary artery disease du to longstanding increased pulmonary blood flow, the therapeutic implicatio are probably the same Since the essential physiologic abnormality is increase resistance to pulmonary blood flow rather than increased volume flow itsel

closure of the patent ductus at this stage can not be expected to be helpful On the contrary, it may be and frequently is disastrous and therefore contraindicated

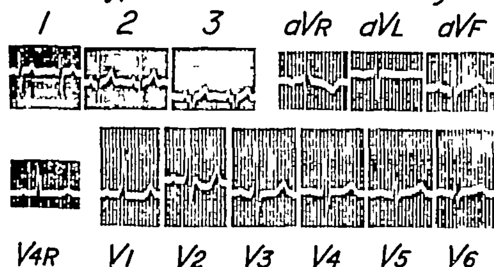


Fig 4 The electrocardiogram in the same patient as in Fig 3 The unipolar precordial leads display typical evidence of uncomplicated right ventricular hypertrophy of the systolic overloading or pressure work variety

SUMMARY

It is recommended that the term "atypical" patent ductus arteriosus be discarded since any patent ductus is typical for whatever situation it represents Furthermore, every clinical syndrome involving a patent ductus should be recognizable and completely evaluated for proper management, using any or all of the available diagnostic methods In this regard, it is not sufficient and in fact may be seriously misleading to include in a single category 'patent ductus arteriosus with pulmonary hypertension This nomenclature actually includes two important syndromes which differ significantly as to physiopathology, diagnostic criteria and surgical management. Details of differential diagnosis in these various *typical* situations involving a patent ductus are outlined



Question Would you describe the diagnosis and treatment of infantile coarctation, with the ductus emptying below the coarctation?"

Answer I don t know what the definition of 'infantile coarctation is, except that it is physiologically a coarctation with the patent ductus entering the aorta distal to the coarctation.

We have pursued the policy of early surgery in these cases and have been very happy to be able both to divide the ductus and resect the coarctation with a primary end to end anastomosis of the aortic ends, and

I think we have a growing feeling that the earlier this is done, the better These children get into trouble early, as most of you know If the surgery is done early there appears to be less likelihood of cardiac asystole upon closure of the ductus. Furthermore, it is a little easier for the surgeon to do a primary resection of the coarctation, even though there might be some considerable length to the hypoplastic or coarcted segment of the aorta.



DISCUSSION

Emile Holman (*San Francisco*)

It is obvious that if you operate on a patient with a very well developed right ventricle, and close the patent ductus, you will get into trouble.

The right ventricle hypertrophies increasingly as time goes on. Obviously, then, one should try to make the diagnosis early and be able to operate in the early years—the first two years of life. I think if one tries to do it beyond that age one will get into difficulty.

Actually, Dr Gerbode, in our clinic, has had such a case I have had no instance in which I have operated when I thought there was any great right ventricular hypertrophy.

Frank Gerbode (*San Francisco*)

We have had about a dozen patients with pulmonary hypertension and patent ductus upon whom we have operated at Stanford, and about five of them had so-called balanced ductus, that is, the pressure by catheter was about the same on both sides of the system, the pulmonary artery and aortic pressures.

I have been able to close the ductus in each instance, but before doing so we have watched the pressures in the pulmonary artery very carefully to be sure they would not rise higher than they were with the ductus open

All of these patients have continued to have some evidence of pulmonary hypertension They have a loud P_2, they have a Graham Steell murmur, and I think possibly some of them may get into difficulty in the future.

We have one adult who had a balanced ductus, who had marked arterio-sclerotic changes in her lungs, and who was in heart failure twice before we operated on her Since the closure of the ductus she has gotten along extremely well, and her pulmonary artery pressure has decreased

We have had one patient with a clear-cut reverse ductus. We tried to close the ductus The pulmonary artery pressure went considerably above the systemic pressure, and we desisted

William H. Muller, Jr. (*Charlottesville, Virginia*)

We feel that the reverse ductus with a high pulmonary resistance and with a pulmonary artery pressure which exceeds the aortic pressure consistently or constantly probably should not be closed It is the reverse ductus in which

there is reversal only during periods of exercise, which is not constant, which can be closed safely

We think one should consider very carefully this latter group When one explores the patient it is important to occlude the ductus for twenty minutes or so, and see how the right heart will tolerate the occlusion, and it is also very helpful to have needles or catheters in the pulmonary artery and aorta during this period of occlusion and observe changes in pressure in the pulmonary artery If this pressure rises markedly and remains high, then it is likely that one will not be able to close the ductus

Rodolfo Kreutzer (*Buenos Aires*)

I would like to point out that the absence of the continuous murmur in the atypical ductus cannot always be explained on the basis of a lack of pressure gradient between the aorta and the pulmonary artery In one of our cases the pulmonary pressure was 73/54, and the aortic pressure was 110/70, and there was only a systolic murmur In another case the pulmonary pressure was 64/41 and the aortic pressure was 110/70, and this patient had a continuous murmur

We think that the way the ductus is implanted is another important factor When the direction is perpendicular to that of the aorta, there is an increased turbulence and the probability of the production of a continuous murmur is greater When the angle favors the flow of the blood, that probability diminishes

EVALUATION OF TRANSPOSITION OPERATIONS

RICHARD J BING (*Birmingham*)—CHAIRMAN

EVALUATION OF TRANSPOSITION OPERATIONS

W. T. MUSTARD (*Toronto*)

Transposition of the great vessels is the leading cause of death due to congenital cardiac anomalies in infancy. It is also the commonest of cardiovascular causes of persistent cyanosis in the newborn.

The condition is now well known and the diagnosis is relatively certain. We shall confine our comments to those cases of complete transposition in an architectural sense; the aorta arises from the right ventricle, the pulmonary artery from the left and there are no gross associated cardiac anomalies. We shall refer to this anomaly as simple transposition. It is recognized, of course, that there must be some mixing of blood for the child to survive birth, and the presence of a small interventricular septal defect, patent foramen ovale or patent ductus would not contraindicate surgical correction.

First attempts at surgical correction of this defect were made by Blalock[1] and his group at Johns Hopkins Hospital, these operations consisted of further mixing of venous and arterial blood between atria and ventricles. The elegant operation devised by Blalock and Hanlon of creating an interatrial septal defect followed by systemic-pulmonic anastomosis to permit further oxygenating of blood met with temporary success However, as recognized by Blalock, this was simply a start and probably overloaded the pulmonary flow.

This was followed by the report of Lillehei and Varco[2] who attempted heroic operations upon the venous side The underlying principle is to transpose the pulmonary venous drainage to the right atrium and the systemic venous return to the left atrium The advantage of this type of operation, as pointed out by the authors, is that the coronary vessels which arise from the aorta, receiving unoxygenated blood, then receive oxygenated blood. These attempts have been partially successful and proved to be at least technically possible.

In evaluating this procedure one has to consider a number of factors Clamping the inferior venae cavae of dogs demonstrates a marked fall in blood pressure in a normal animal which would make it extremely hazardous in a dangerously ill patient The superior vena cava can be clamped with impunity and one may remove the superior vena cava and transpose it to the left atrium. The success of this operation depends upon the completeness of the venous transposition To transpose the right pulmonary veins to the right atrium and the superior vena cava to the left atrium accomplishes one quarter

of the effect desired Transposing the pulmonary veins on the right side to the right atrium and at the same time transposing the inferior vena cava to the divided cardiac end of the pulmonary veins provides perhaps two-thirds correction The authors experienced a number of difficulties, the greatest of which was to get proper balance in venous flow to the heart Opening one anastomosis before another creates gross imbalance. This problem can be overcome with the use of an extracorporeal circuit and we have attempted this procedure on three occasions with no success

The main criticism of transposition on the venous side is that under ideal conditions with either hypothermia or an extracorporeal circuit one can accomplish only about 75 per cent correction. One would always have the problem of the pulmonary venous drainage from the left lung entering the wrong atrium, perhaps this could be overcome at a later stage with a graft Furthermore the coronary sinus drainage would persist on the wrong side It is our feeling at the present time that operations on the venous side should be pursued in the hope that one may permit these babies to survive for further surgery

If we turn our attention to the arterial side it would seem obvious to transpose the great arterial trunks, if these are the same diameter, which occurs in about 75 per cent of patients with simple transposition. Bailey et al [3] have performed this operation on the human with technical success For vessels of unequal size Bjork[4] has performed cross grafting division and resuture and has demonstrated this is practical from an experimental point of view This author has pointed out that retransposing the great vessels is impractical with low pulmonary pressure. In simple transposition the pressure is always high in the pulmonary artery and would be low only in the presence of another gross cardiac anomaly

Cross et al.[5] have used temporary shunts in experiments in which the great vessels were clamped

However, the problem is not as simple as it appears because these procedures would leave the coronary arteries on the side of the unoxygenated outflow While it is true that certain cases of tricuspid atresia and tetralogy of Fallot survive with the same degree of desaturation of the coronary flow, their life expectancy is short. Operations ignoring the coronary flow could only be palliative in cases of simple transposition

We have recognized this difficulty and have attempted to transpose the great arterial trunks to include the left coronary vessel. In transposition of the vessels the left coronary artery arises somewhat higher and more anterior from the aorta than normal and lends itself to retransposition of the great systemic trunks The right coronary arising from the pulmonary artery is compatible with longevity and we did not feel it necessary to transpose both coronary vessels In order to accomplish this one requires an extracorporeal circuit, and as yet there have been no successes [6] Transposing the left coronary artery to a systemic vessel is feasible, and we have performed this on one occasion with the coronary arising from the pulmonary artery This then would leave one with retransposition of the great vessels themselves after systemic-coronary anastomosis as another approach. These possibilities are

being explored at the present time Another problem as yet unexplored is the interference with the nerve supply to the heart.

SUMMARY OF TYPES OF OPERATION

1. Increased mixing of the blood-flow by creation of an interatrial septal defect and systemic-pulmonary anastomosis This operation should be given up in simple transpositions because the shunt is from right to left.

2 Partial correction by anastomosis of the superior vena cava to the left atrium and the right pulmonary veins to the right atrium

3. The right superior and inferior pulmonary veins to the right atrium and the inferior vena cava to the left atrium.

4. The superior vena cava to the left atrium, right pulmonary veins to the right atrium and inferior vena cava to the left atrium

On the arterial side one has the problem simply of not the retransposition of the great arterial trunks but also of one or two coronary vessels.

The systemic anastomosis of a coronary vessel is not impractical, particularly in transposition, as it rises higher and more anterior than it does in normal hearts, and retransposition of the arterial trunks is feasible

CONCLUSION

It is our opinion that at the present time the approach to correction, that is, complete correction of simple transposition of the great vessels, lies on the arterial side and this can be accomplished by the use of hypothermia or an extracorporeal circulation

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DISCUSSION

Clarence Crafoord (*Stockholm*)

I have had no experience because I have not done a case of transposition. When one of my men was here a year ago he tried in different ways to correct the condition in the experimental laboratory, and he also tried one clinical case in the operating room when I assisted him.

The principle on which he has been working has been that it is possible

to dissect both the aorta and the pulmonary artery all the way down into the ventricles, so that you come below the valvular level.

If there is not a ventricular septal defect, one can be created, or if there is a ventricular septal defect, the aorta can be turned over and sutured into the ventricular septal defect Then the pulmonary artery is sutured into the opening of the right ventricle from where the aorta has been cut out. By that method both the coronary arteries are turned, together with the aorta, onto the left ventricle, and the pulmonary artery arises from the right ventricle.

It sounds complicated, but when it is performed by someone who has been trained to do it in the laboratory, it is an operation that can be performed in about half an hour

In the case where we tried it, it was done under hypothermia, with complete occlusion of both the cava, the azygos vein and the pulmonary artery and the aorta. We could bring back heart action for a short period of time, but there was a complete block. The suturing in that case unfortunately also touched on the bundle of His If one can avoid the bundle of His and get the vessels into the right position without any trouble from the conduction system, it could be a possibility It is possible to do this in animals and have them survive for a short period of time.

Earle B Kay (*Cleveland*)

Last year Dr Cross and I presented a paper before the American Thoracic Association on the use of shunts and the surgical treatment of transposition of the large vessels This technique was employed in 3 patients without success Unfortunately, as you know, these are extremely serious risks By the time the plastic extracardiac shunts were placed in order to allow us to divide and retranspose the vessels, the hearts invariably failed and in no instance were we able to complete the operative procedure. We concluded that that particular technique was of no value in the treatment of transpositions

During the past year we have operated upon 3 infants with transposition of the large vessels by a technique which we believe may have merit. These 3 children varied from 4 week to 3 months of age. The technique employed consisted first of transecting the right pulmonary artery adjacent to the pulmonary conus The proximal end was closed, the distal end of the right pulmonary artery was anastomosed to the aorta coming from the right ventricle. This anastomosis was then opened and allowed the blood circulating from the right ventricle to go both to the right lung and to the aortic arch. A large anastomosis was then made between the pulmonary conus and the arch of the aorta just distal to the innominate artery This anastomosis was then opened, which in turn allowed blood to circulate both to the left lung and the arch of the aorta from the left ventricle. The aortic arch was then transected proximal to the innominate artery, the two ends being closed. Blood now circulated from the right ventricle to the right lung and from the left ventricle to the arch of the aorta and to the left lung The left pulmonary artery was then transected adjacent to the conus, the proximal end closed, and the distal end anastomosed by means of a homologous graft to the aorta

arising from the right ventricle. At the conclusion of this anaston transposition had been completely retransposed

Since the arterial oxygenation of the patients is markedly reduc beneficial to give oxygenated transfusions during the course of the o In 2 patients the technique as described was completed. The patie markedly improved afterwards, but died during the postoperative per five hours postoperatively, prior to which time the baby had respon and his color was excellent Cause of death could not be ascertained autopsy examination. In a second child the operation was staged rat being completed in one operation. Unfortunately, this particular c had an undetected coarctation of the aorta so that the entire aortic blood was shunted into the left lung This patient died from hem pneumonitis The third patient, a 4-week-old infant, tolerated the o extremely well but died approximately five to ten minutes after t was closed from what was found to be kinking of the homologous gr closure of the chest incision. In the open chest the length of the g peared to be optimum but in closing the chest incision, a somewl homologous graft kinked the pulmonary artery. This is a technical fac can easily be eliminated in the future.

As yet no attempt has been made to transpose the coronary arterie the preoperative oxygen determination in the arteries in patients wit position of the vessels usually is between 20 to 35 volumes per cent a the oxygen content of venous blood in a normal individual is betwe 80 volumes per cent, it is felt that after the vessels are retranspo increased oxygen content of the coronary arteries from that noted p tively should be sufficiently beneficial that transposition of the c arteries as well may not be necessary

Sanford E. Leeds (*San Francisco*)

We have some experimental evidence on the relation of the septal whether atrial or intraventricular, to the survival of dogs after exper transposition

We used a method similar to that described by Blalock and Hanl anastomosed the proximal end of the azygos vein to the distal end right superior pulmonary vein, thereby draining the upper part of t lung into the right side of the heart

The right pulmonary artery was connected directly to the aorta end to end anastomosis with the innominate or brachiocephalic arte the proximal end of the right pulmonary artery was ligated.

At a later operation the upper lobe of the left lung was excised T done to favor the right to left shunt through the interatrial septal defec was made in half of the animals The only output of the right ventric was to a branch of the left pulmonary artery to the lower lobe. Th lung was supplied from the aortic arch We produced complete pul atresia by removing the remaining left lower lobe

We did this in dogs with and without interatrial septal defects. I was no interatrial septal defect, ventricular fibrillation came on imme

upon clamping of the left pulmonary artery If there was a septal defect, however, the dogs were able to live up to 6 hours

We plan to repeat this procedure with interventricular septal defect, feeling that anatomically we are apt to get a better right to left shunt which may permit the animals to survive

MITRAL STENOSIS AND MITRAL INSUFFICIENCY

E. COWLES ANDRUS (*Baltimore*)
EGBERT H. FELL (*Chicago*)
} CHAIRMEN

PHYSIOLOGIC STUDIES IN MITRAL VALVULAR DISEASE

RÉJANE M. HARVEY (*New York City*)

Circulatory dysfunction in rheumatic heart disease has long been recognized as springing from at least two main sources, the mechanical difficulties imposed by valvular lesions and the insufficiency of the myocardium itself. This insufficiency may result from longstanding strain inflicted on the cardiac muscle by altered valvular function or, independently of mechanical cause, may occur consequent to intrinsic myocardial damage from the rheumatic process Our understanding of cardiac function in rheumatic patients would be increased if one could separate the mechanical from the myocardial components in order to investigate further the disability these subjects experience The modern surgical approach to rheumatic mitral stenosis introduced by Drs Bailey and Harken, attacking as it does only the mechanical features of valvular lesion, affords an opportunity to study this problem Furthermore, if it can be shown that myocardial insufficiency exists as a separate dysfunction, it behooves the physician and surgeon to be certain that the prospective candidate for mitral commissurotomy is suffering from a predominantly mechanical lesion, namely block at the mitral valve, and not chiefly from myocardial insufficiency

As a result of cardiac catheterization studies made in a series of patients who were being considered for mitral surgery, three groups of individuals have emerged In the first two groups, whose disability was undoubted, clinical and physiologic findings have led us to the conclusion that in one the *predominant* difficulty was due to mechanical block, while in the second it resulted from myocardial insufficiency. In the third group no physiologic abnormalities could be demonstrated and therefore their symptoms could not be ascribed to heart disease.

MITRAL STENOSIS WITH MITRAL BLOCK

The hemodynamic pattern of the patient suffering from mitral stenosis and significant mitral block has been amply described, as have been some of the effects of surgery upon such patients These findings are illustrated in Fig. 1. This young man with a progressive history of dyspnea unrelieved by digitalization was free of manifestations of cardiac failure He had a reduced cardiac output, pulmonary hypertension and a normal right ventricular diastolic pres-

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sure. As you know, elevation of the right ventricular diastolic pressure in acquired heart disease is the earmark of right-sided heart failure On exertion we find a fixation of the cardiac output Pulmonary artery pressures rise, presumably as a result of increase in heart rate as stressed by Dr Harken and his co-authors, in face of an increase in right ventricular ejection and a rise in right ventricular diastolic pressure, certainly an indication that this chamber was under strain One month following commissurotomy, the striking change is a reduction in the level of pulmonary artery pressures at rest and during exercise. Eleven months later, when the patient was once more gainfully employed, we find a further decrease in the resting level of pulmonary artery

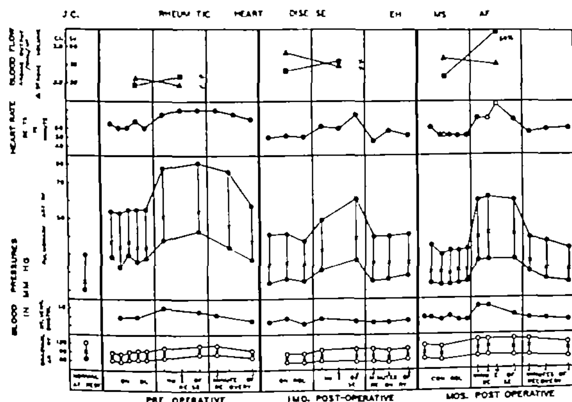


Fig. 1 Graphic representation of hemodynamic data in a patient before and after mitral commissurotomy (From Circulation vol. 7 1953)

pressures Furthermore, on exercise the cardiac output now increases normally Despite this greater flow through the pulmonary bed during exercise the pulmonary pressures are not higher than preoperatively

The hemodynamic pattern of rheumatic mitral stenosis with block is a reduced cardiac output which is fixed in exertion, pulmonary hypertension which is augmented on exercise, and normal right ventricular diastolic pressure which may become elevated during stress Following surgery and relief of mitral valvular block, we find only occasionally an increase in output at rest. More usually the same blood flow is noted at rest postoperatively However, on exertion the cardiac output rises more adequately Pulmonary hypertension is ameliorated but pulmonary pressures are rarely returned to normal.

That these pressures can be returned to normal is illustrated by the findings seen in the second patient (Fig 2) We see much the same hemodynamic picture preoperatively as was shown in Fig 1 Again following surgery there

is a progressive fall in pulmonary artery pressures without significant change in the resting level of cardiac output However, at one year postoperatively the pulmonary pressures are normal both at rest and during exercise.

It might be thought, therefore, that a demonstration of pulmonary hypertension in the presence of symptoms would suffice to diagnose significant mitral block It should be stressed that cardiac symptoms and pulmonary hypertension in the rheumatic patient with mitral stenosis can be also due to left ventricular failure

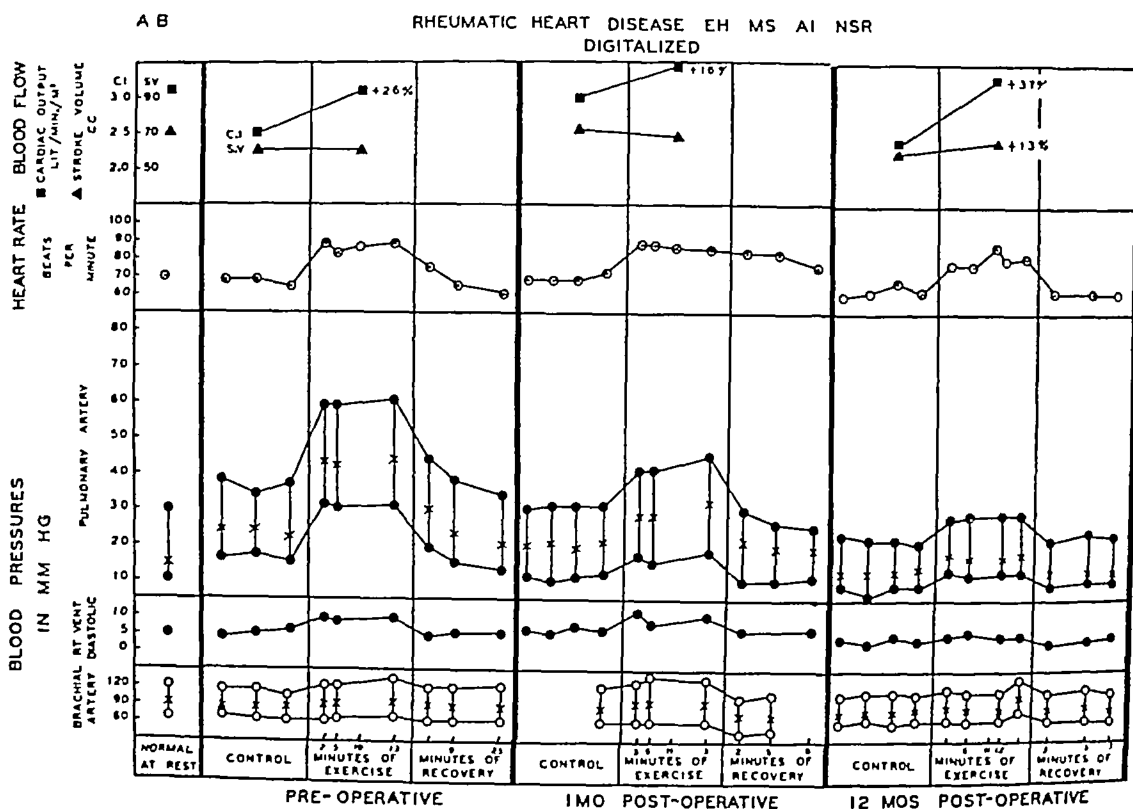


Fig 2 Graphic representation of hemodynamic date in a patient before and after mitral commissurotomy (From Circulation, vol 11, 1955)

This is illustrated in Fig 3. This patient with the murmurs of mitral stenosis, mitral insufficiency and a basal diastolic murmur was dyspneic and orthopneic at the time of study. We have much the same hemodynamic pattern here that we found in the previous two cases presented, namely, a reduced cardiac output and pulmonary hypertension Following the intravenous administration of Digoxin we find a rise in cardiac output and a fall to normal in pulmonary artery pressures as the failing left ventricle empties better and mobilizes blood from the pulmonary vascular bed

The patient with pulmonary hypertension resulting solely from mitral stenosis and significant block does not react to digitalis bodies and a medical regimen in this fashion, as is seen in Fig 4. Here are the hemodynamic data of a young man with a reduced cardiac output and severe pulmonary hypertension who was studied following acute digitalization The cardiac output did not rise appreciably nor did pulmonary artery pressures change. The patient was restudied two weeks later, following complete digitalization and the institution of a more rigorous medical regimen. There were no changes in resting

hemodynamics, and exercise produced an inadequate rise in cardiac output and a more severe pulmonary hypertension. One month following commissurotomy, the pulmonary pressures are almost normal at rest and during exercise and blood flow increases satisfactorily on exertion

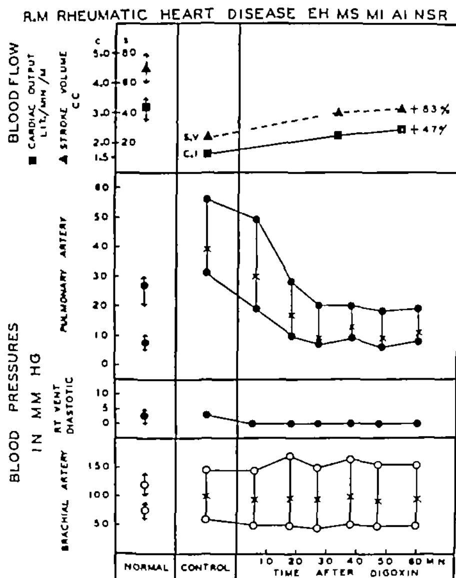


Fig. 3 Graphic representation of hemodynamic data in a patient with rheumatic heart disease before and after the intravenous injection of Digoxin. (From Circulation vol. 7, 1949)

MITRAL STENOSIS WITH MYOCARDIAL INSUFFICIENCY

The previously described findings in mitral block are more or less as expected. However, in our study of mitral stenosis, another group of patients was identified in whom the physiologic dysfunction was entirely different. In this group with mitral stenosis myocardial insufficiency and not mechanical block limited their performance. Fortunately their physiologic findings clearly separated them from the group with mitral block. This separation is a necessity since their symptoms are as severe as those with mitral block and hence they may seek surgery Although the history that these patients give does not always identify them, we have found that frequently their cardiac symptoms

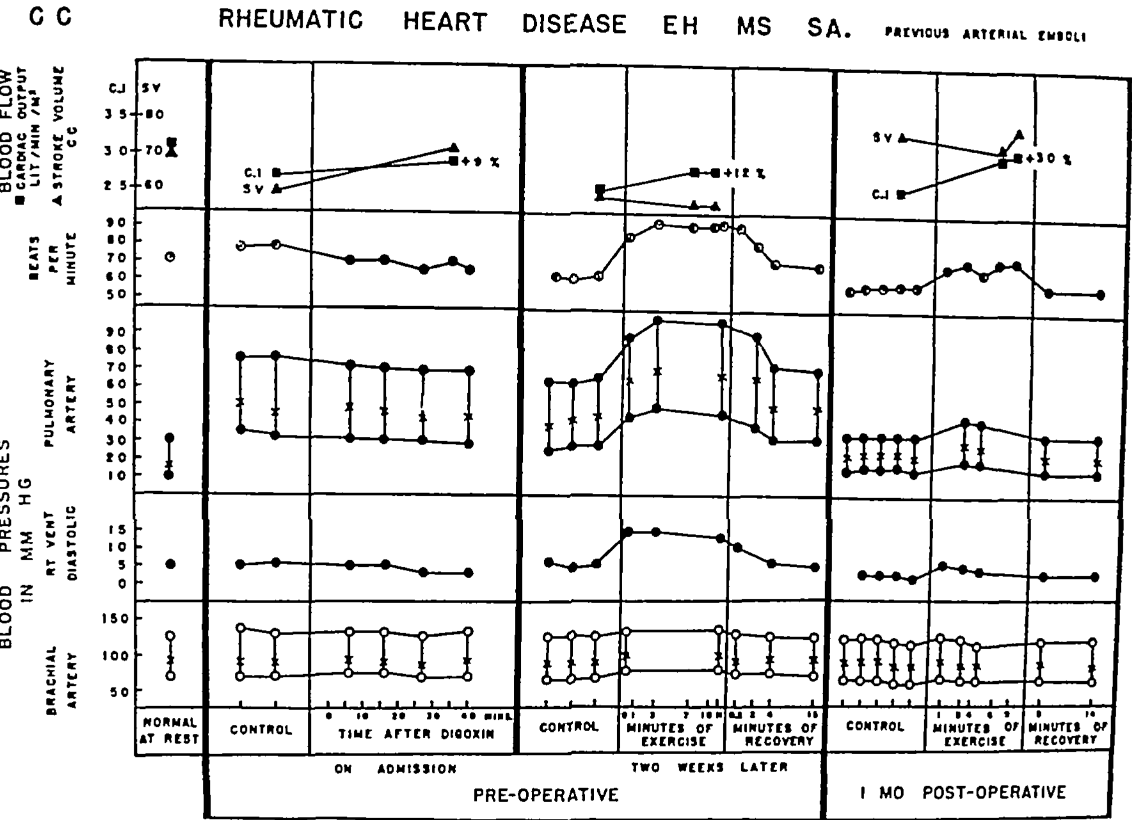


Fig. 4 Graphic representation of hemodynamic data in a patient with rheumatic heart disease which indicates no amelioration of pulmonary hypertension with Digoxin but definite reduction in this pressure following commissurotomy (From Bull N Y Acad Med, vol 30, 1954)

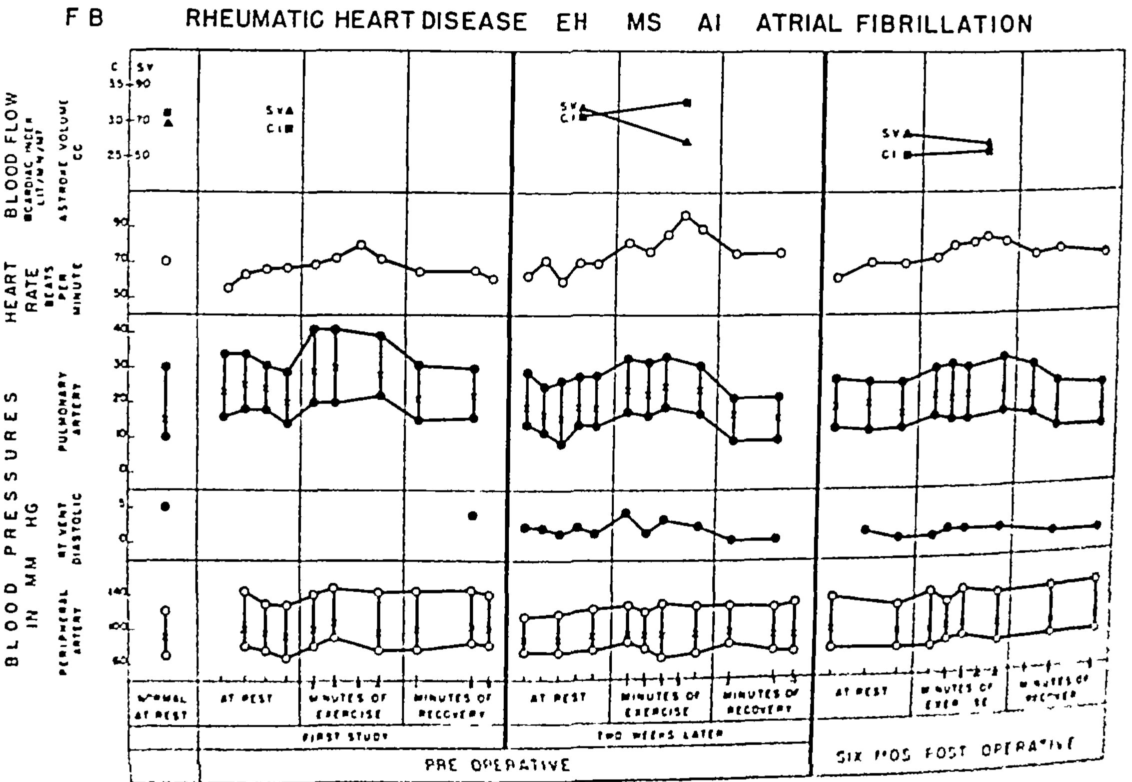


Fig 5 Graphic representation of hemodynamic data in a patient before and after mitral commissurotomy (From Circulation, vol 11, 1955)

are episodic, amenable to medical therapy, and disability is not progressive and unremitting in contrast to those subjects with mitral block.

This group is well exemplified by the findings in a young handy man (Fig 5) whose intermittent symptoms, which included bouts of severe heart failure, were greatly ameliorated by digitalis At the time of the first study he had no clinical signs of pulmonary or peripheral congestion. The cardiac output was normal at rest and the pulmonary artery pressures, which were slightly increased, rose further on exercise. After two weeks of bed rest in the hospital a second catheterization was done. During this interval the only clinical change was a distinct decrease in heart size (Fig 6) On this second evaluation the cardiac output at rest was the same as on the first measurement, but with the same ventricular rate, there had been a small but definite fall in the pulmonary artery pressures Comparison of the resting hemodynamics in these two preoperative studies (Fig. 5) reveals that the change on medical management is best explained as due to a decrease in left ventricular myocardial failure. This assumption is strengthened by the response to the exercise performance made on this second study Although the patient reached

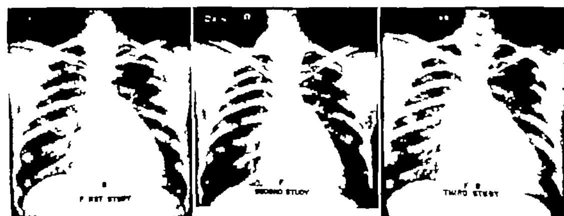


Fig 6 Roentgenograms of patient F B taken at the time of his first second, and third cardiac catheterizations. (From Circulation, vol. 11 1955)

the same level of oxygen consumption during effort on both occasions, the second time, despite a ventricular rate which was even higher than in the first exercise period, there was no significant rise in pulmonary artery pressures as there had been when the mild left ventricular failure still existed. The cardiac output did not increase significantly on exercise. It was recognized that this patient's resting hemodynamics and his response to exertion, namely a fixed cardiac output without rise in pulmonary artery pressures, were quite different from those of patients with significant block. The absence of real pulmonary hypertension particularly was at variance with the usual experience in severe mitral valvular stenosis

Since one could not, in view of our lack of knowledge, be certain if mitral stenosis in its mechanical aspects were responsible for the abortive response in cardiac output during exercise, and in view of his history of repeated disability, this patient was permitted to undergo mitral commissurotomy The orifice was somewhat narrowed and the edges split easily For five months

after operation he was in much the same state as preoperatively when he was out of congestive failure with a well controlled ventricular rate. However, following an upper respiratory infection, he once more went into congestive failure with an increase in heart size. After bed rest and diuretics the heart size again decreased, but at the time of his third catheterization, when all signs of congestion had disappeared, it was still larger than preoperatively (Fig. 6). At this time, six months postoperatively, the cardiac output both at rest and during exercise was 1 liter lower than preoperatively and the blood flow was still fixed during exercise. The pulmonary artery and right ventricular pressures were the same as on the second study. They increased only

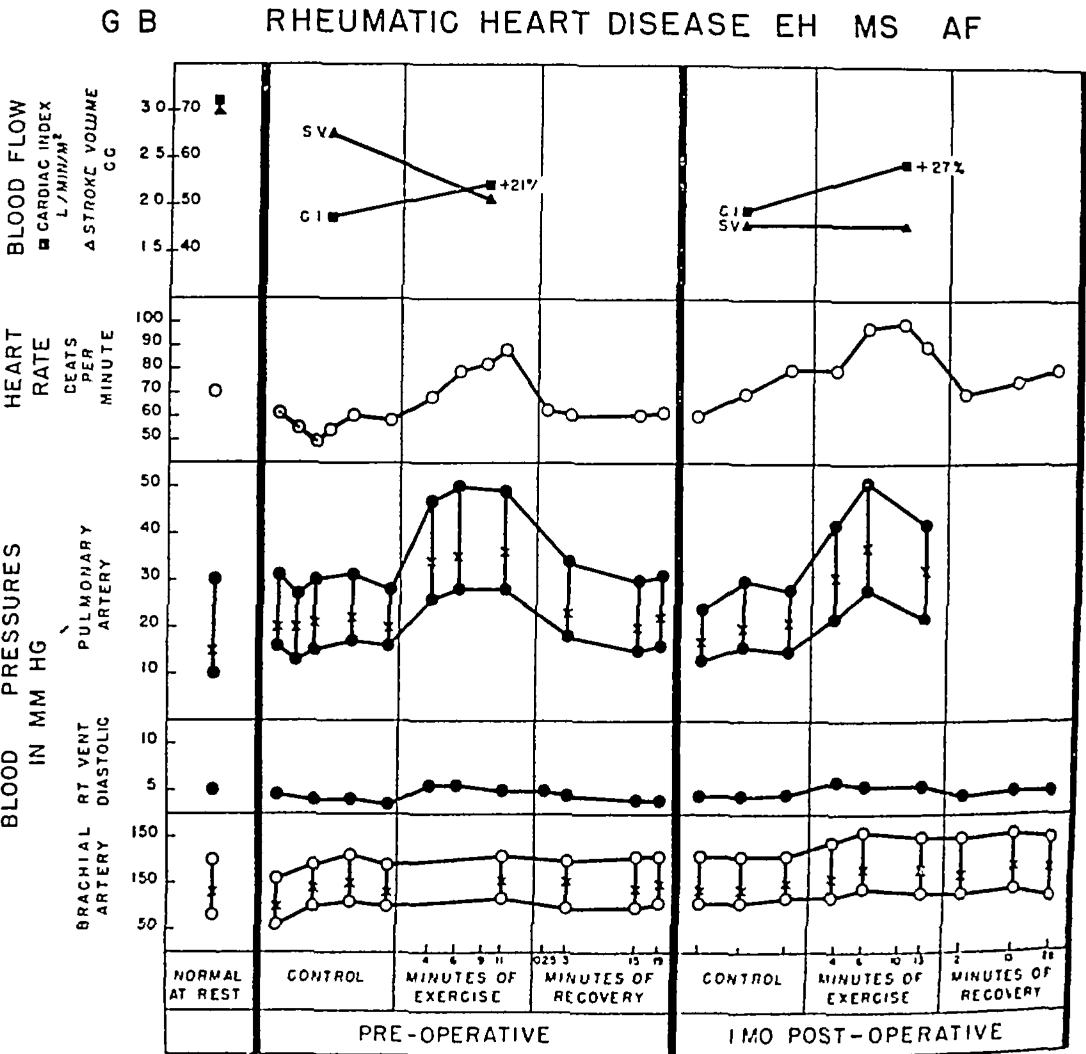


Fig 7. Graphic representation of hemodynamic data in a patient before and after mitral commissurotomy. (From Circulation, vol 11, 1955)

very slightly during exercise, as was the case during the second study. The operative intervention then had produced no improvement in hemodynamic function. Moreover, the level of blood flow was now below normal, probably as a result of the direct and continuing effect of the rheumatic process on the myocardium. It is therefore concluded that mitral block was not the primary cause of dysfunction in this man.

Figure 7 also illustrates the hemodynamic picture which may be encountered in this group of patients who suffer predominantly from myocardial

insufficiency This 39-year-old salesman complained chiefly of fatigue and to a lesser extent of dyspnea. Ankle edema had been relieved by digitalization He was found to have a very low cardiac output at rest, and minimal pulmonary hypertension. On exercise, despite a subnormal rise in cardiac output, pulmonary artery pressures rose to moderately hypertensive levels Although the resting pressure in the pulmonary artery was only slightly elevated, the exercise hypertension was disturbing, as it could be ascribed either to some degree of mitral block, which only became important when blood flow increased, or to left ventricular failure which appeared on exertion. Since his symptoms and exercise hypertension could be due to mitral block, commissurotomy was done and the surgeon widened a narrowed valve orifice. The postoperative catheterization at one month showed no change over the preoperative performance at rest or during the same degree of exercise. Three years and three months have passed since surgery and there is no evidence of clinical improvement.

Of a group of 60 patients who have been considered for mitral surgery, of whom 45 have been catheterized, we have found 8 patients in whom the physiologic derangements are exemplified by the 2 patients just presented. The other 6 were not offered surgery

All 8 of these subjects had moderate to severe cardiac symptoms which were episodic in their occurrence, and in all, when evidence of cardiac failure was abolished by a medical regimen, pulmonary hypertension was minimal or absent at rest. In the 2 who underwent surgery there was neither clinical nor physiologic evidence of improvement after commissurotomy If one accepts the experience encountered in the first group as characteristic of mitral block, a state which was expressed hemodynamically by pulmonary hypertension at rest, which was aggravated by exertion, and which yielded to valve fracture as attested to not only by clinical improvement, but also by objective measurements of a decrease in lesser circuit pressures postoperatively, then the patients in this second group do not have appreciable mitral valve block. The subnormal response in cardiac output during exercise, be it normal or low at rest, was common to both groups and hence does not help in differentiating them.

If one does not accept the deranged dynamics in these individuals in the second group as chiefly the result of mitral block, one must attempt a further explanation of their difficulties All are victims of rheumatic heart disease and hence it is likely that myocardial lesions exist in them as well as valvular cicatrices Although mitral valvular damage has occurred as indicated by auscultation, there appears to be little hemodynamic evidence of obstruction to blood flow at rest at the mitral valve in these patients with almost normal lesser circulation pressures In none of these individuals, however, does the cardiac output respond normally to the demands of exertion. This suggests an insufficiency in myocardial performance which is not related to mechanical obstruction within the circulatory channels Indeed, this insufficiency is probably the major circulatory defect of the patients in the second group

The importance of differentiating the group with mitral block from that with predominantly myocardial insufficiency, and the catheterization data appear to make this a feasible differentiation, is obvious when one considers

offering surgery to any patient with mitral stenosis. It would seem only logical to insist on a demonstration of pulmonary hypertension in each prospective candidate in order to avoid selecting one with predominantly myocardial insufficiency.

ASYMPTOMATIC GROUP

The abnormal dynamic state which is considered to have as its basic mechanism poor myocardial function, is not to be confused with the state of

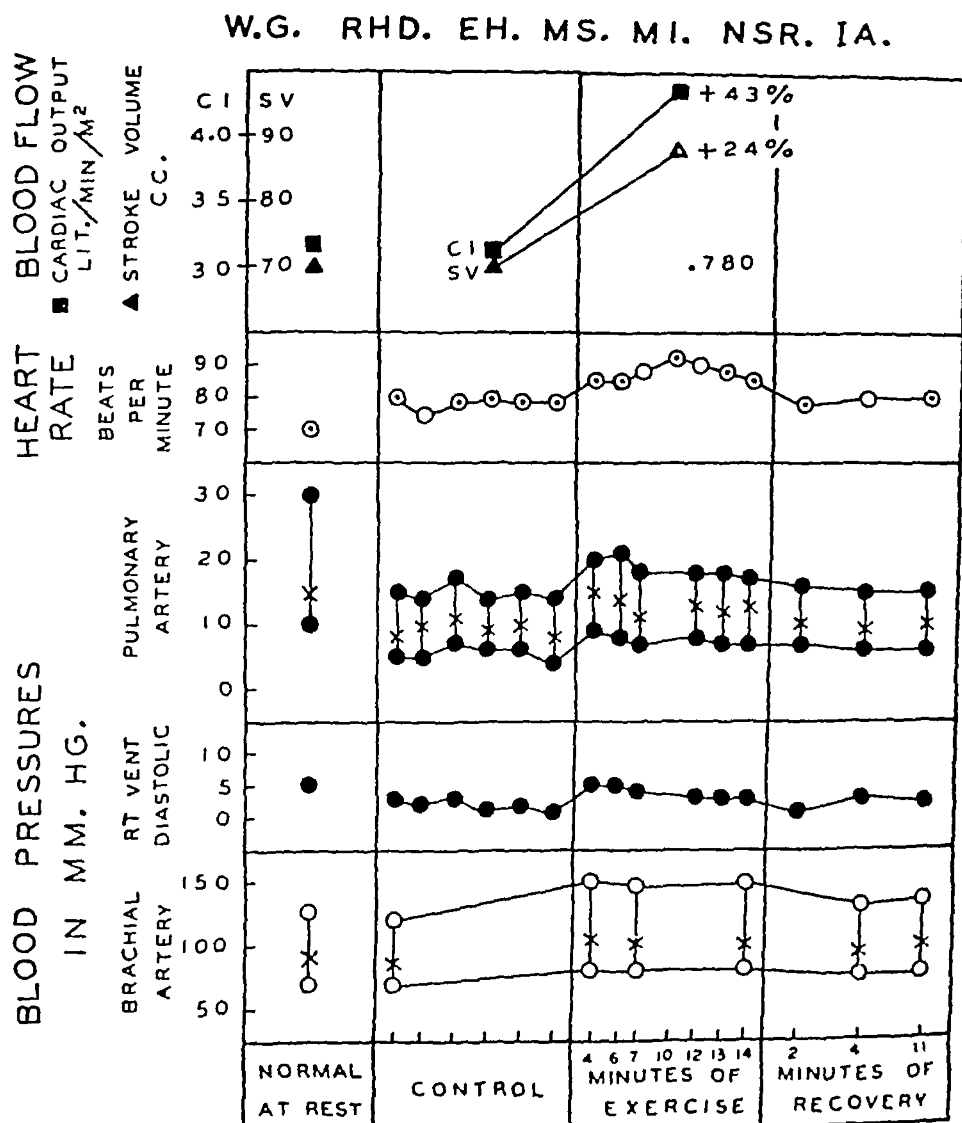


Fig 8 Graphic representation of hemodynamic data in a patient with rheumatic heart disease at rest and during exercise (From Circulation, vol 6, 1952)

certain other patients with mitral stenosis who also do not have pulmonary artery hypertension. These latter subjects are either asymptomatic entirely or have symptoms which are noncardiac and often are iatrogenic. Studies in these individuals have revealed a normal level of cardiac output and a normal response of this function on exertion.

The findings in a young electrician illustrate this final group. This patient was asymptomatic except for hemoptysis associated with respiratory infec-

tions. Physiologic studies revealed no abnormalities either at rest or during exertion (Fig. 8). It seems unlikely that disordered cardiac function was the basis of this patient's hemoptysis.

SUMMARY

Physiologic studies have clarified our understanding of the patient with mitral stenosis. The postulate that significant block at the mitral valve produces pulmonary hypertension which can be alleviated by surgery has been confirmed.

It has further been shown that there exists a group of patients with disabling symptoms, in whom myocardial insufficiency is the predominant lesion. This group is characterized by little or no pulmonary hypertension and will not benefit from surgery.

Finally, there is a group of patients with auscultatory signs of mitral stenosis in whom the circulation is not deranged.

DIAGNOSIS OF MITRAL STENOSIS BY ELECTROKYMOGRAPHY OF THE LEFT ATRIUM

OLGA M. HARING, HERBERT D. TRACE AND ALDO A. LUISADA (*Chicago*)

There are two sets of indications for intervention in cases with mitral valvular lesions. One set is for mitral stenosis and is more liberal, while the other set is for mitral insufficiency and is used only when the patient is definitely incapacitated. The difficulty arises from the problem of making an exact diagnosis. Is there a pure stenosis? Is there an insufficiency? Or is there a mixed stenosis and insufficiency?

Early in our work at Mount Sinai Hospital in Chicago, we found ourselves in frequent disagreement about the degree of insufficiency that a diseased mitral valve might have. Four specialists were involved in this disagreement—the clinical cardiologist, the cardiac physiologist, the surgeon and the pathologist The pathologist, by reason of tradition, usually carried the most weight. The surgeon, because of increasing self-confidence in his ability, was talking more and more about the "diagnostic finger" and frequently was expressing opinions which were in opposition to the others. The clinician, having been repeatedly overruled on this particular point, was often quiet and attentive but did not refer patients for cardiac surgery So it remained for the cardiac physiologist to resolve these difficulties.

We studied these patients by means of electrokymograms of the left atrium before and after surgery. It soon became evident that even when the surgeon reported no regurgitation because he could not feel a thrill or a regurgitant jet, the electrokymogram revealed a systolic plateau, indicative of regurgitation. At first we rationalized that many factors, including the operative position of the patient and the decrease of cardiac output due to anesthesia, decrease temporarily the severity of regurgitation. Moreover, the subjective feeling of the "jet" may be at fault Later on, by carefully checking our tracings, we became convinced that, even though the meaning of a plateau is that of regurgitation, there is a difference between the plateau of predominant stenosis and that of predominant insufficiency.

The scheme of electrokymography is shown in Fig. 1.

The *normal left atrial kymogram* (Fig. 2) shows a presystolic negative wave corresponding to the atrial contraction, followed by a systolic negative wave caused by the pull of the left ventricle on the atrioventricular floor during

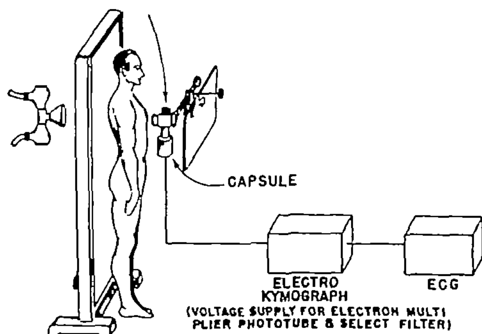


Fig 1 Schema showing the method of recording the electrokymogram. The device in front of the chest expresses the change in the amount of radiation coming through a slit placed over the particular part of the heart under study by change in voltage which is in turn recorded by the writing device in the electrokymograph. The electrocardiogram and the phonocardiogram may be simultaneously recorded.

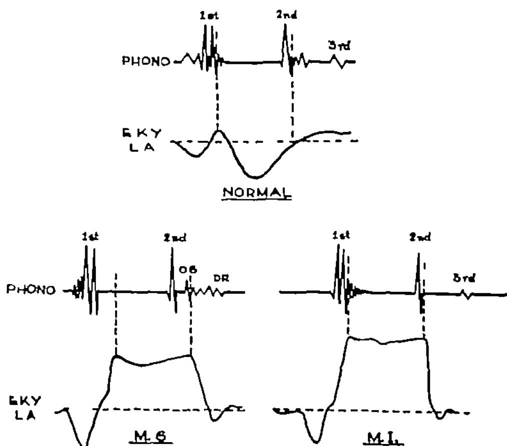


Fig. 2. The electrokymogram of the left atrium in a normal individual one with mitral stenosis (M. S) and one with mitral insufficiency (M. I). See text.

ventricular systole A tracing with a systolic plateau, instead of the normal negative wave, is characteristic of mitral regurgitation, as described by Luisada and Fleischner in 1948.

We now have studied over 100 cases, and in about one-third of them we were able to correlate the various graphic data with the surgical findings We found that although there is a systolic plateau in every case of mitral valve lesion, there is a difference in the onset of the plateau, in its duration, and in the number of projections in which the plateau is present. Arbitrarily we called a "late plateau" one which began at least 0.10 second after the first loud vibration of the first heart sound; we called an "early plateau" one whose onset began less than 0.06 second after the vibration of the first heart sound, and we called an "intermediate plateau" one which started between 0 06 and 0 10 second after the first vibration of the first heart sound We felt that a *late plateau* was definite evidence of predominant stenosis of the mitral valve while an *early plateau* indicated predominant insufficiency.

MATERIAL AND METHODS

Thirty patients were studied, 20 women and 10 men in the age groups from the third to the sixth decade In 25 cases, the clinical diagnosis was that of "pure" mitral stenosis, in 5 others, that of predominant mitral regurgitation with some degree of stenosis. Two patients were not submitted to surgery, 24 underwent mitral commissurotomy, in 2 other patients, following valvular exploration, an atrial appendectomy was done, and in 2, a transventricular mitral suture of the posteromedial commissure, as described by Bailey, was performed

Correlation of the electrokymographic findings was done in 28 cases with surgical findings, in the 2 others, with data obtained at autopsy.

Left atrial electrokymograms were done with the patient in a sitting position, in several projections in the two obliques and in the right lateral, the slit of the phototube was placed at two levels—a higher and a lower. If in the postero-anterior view the atrial appendage could be visualized, a tracing was recorded over this structure Moreover, two densograms of the left atrium were also taken in the right lateral projection.

For timing purposes, a sound tracing was recorded and the film was recorded at a speed of 75 mm/sec in order to analyze correctly the time relationship of the waves A calibrating device was used for evaluation of the amplitude of the waves. The apparatus used were a Sanborn Electrokymograph and a Sanborn Twin-Beam Interpretation of the tracings included the following points

1. Do the tracings, normal or abnormal in configuration, correspond to a left atrial pattern? Arterial, ventricular and mixed patterns should be excluded They are sometimes recorded if the border of the left atrium is not clearly visualized, if the patient changed position, or if superimposition occurred because of nearness and transmitted pulsations of other enlarged chambers or vessels
2. Is there a presystolic wave?
3. Is there a systolic plateau?

4 In how many of the projections does it occur?

5 Is the plateau early, intermediate or late?

Correlations were made between the number of projections and the number of tracings with the existence of a plateau and its onset and end. This permitted drawing certain diagnostic conclusions, such as mitral stenosis with moderate or with marked regurgitation, and mitral regurgitation with moderate or marked stenosis

RESULTS AND COMMENTS

In all 30 cases of mitral valvular disease, a systolic plateau was found in one or several projections of the left atrium. The plateau was of the early type in 4 patients, intermediate in 4 others, and late in the remaining 22 (Table 1)

In 2 of the 4 cases with an early plateau, this plateau was present in every projection of the left atrium. In the other 2 cases, the plateau was present in several projections Marked regurgitation was confirmed in 3 patients by

TABLE 1

	Projections with Plateau	*Late Plateau*	*Intermediate Plateau*	*Early Plateau*	*Regurgi-tation*	*Calcifi-cation*
1	++	X			–	
2	++	X			–	
3	++	X			–	
4	+++	X			–	
5	++	X			–	
6	+++		X		++	
7	++	X			–	X
8	++	X			–	X
9	+	X			–	X
10	++	X			–	
11	++	X			+	X
12	++	X			–	
13	+	X			–	
14	++	X			–	
15	++	X			+	X
16	++	X			–	
17	++	X			+	X
18	++			X	+++	
19	+++			X	+++	X
20	+++			X	+++	X
21	++	X			+	X
22	++		X		+	X
23	++	X			+	X
24	++		X		+	
25	++	X			–	
26	++		X		–	
27	++	X			+	X
28	++	X			–	
29	++			X	+++	
30	++	X			–	

surgery and in one by autopsy. The intermediate plateau was recorded in one case in all the projections and in 3 cases in several projections of the left atrium. Surgery confirmed a moderate amount of regurgitation in one case, some degree of regurgitation in another, while no regurgitant jet was felt in the third case. The late plateau appeared in one case in every projection, in 19 cases it was found in several projections; in the 2 others it was found in only one projection of the left atrium. Surgery confirmed some degree of regurgitation in 6 out of these 22 cases

The results of our study raise several problems:

1. *Is the systolic plateau of the left atrial electrokymogram a true expression of mitral regurgitation?* There has been sufficient proof given by clinical and experimental studies of several groups in the medical literature, that the systolic expansion of the left atrial wall is a result of insufficiency of the mitral valve causing regurgitation of blood during ventricular systole Pressure curves obtained in 1915 by Wiggers from the left atrium, in experimentally produced mitral insufficiency, demonstrate the same pattern. Electrokymographic and intracardiac pressure studies obtained in our laboratory in experimental animals show identical findings in dogs with surgically produced mitral insufficiency.

2. *What causes the delayed onset and end of the plateau in a predominant mitral stenosis?* In mitral stenosis, as opposed to pure mitral insufficiency, the diastolic pressure of the left atrium is high, therefore, regurgitation of blood will start only at that point at which the systolic pressure of the left ventricle reaches a height which is sufficient to overcome the atrial pressure, that is, during the phase of systolic ejection. After the opening of the mitral valve, the ventricular pressure decreases rapidly. However, mitral opening is delayed in comparison with cases without stenosis and the drop in pressure is slower on account of the narrowing of the mitral valve.

3. *What are the diagnostic advantages of serial electrokymography of the left atrium?* Delayed onset and delayed end of a plateau are therefore definite proof of mitral stenosis in the presence of some regurgitation, while an early plateau is evidence of free regurgitation without severe stenosis. A systematic study of the configuration of the plateau in the various projections, improvement of the calibrating methods, and correlation of all the graphic data may lead to a more precise determination of the amount of blood regurgitated, the condition of the left ventricle, and the localization of the lesion

CONCLUSIONS AND SUMMARY

Electrokymography of the left atrium if applied correctly is the simplest and most reliable diagnostic method of recognizing which is the predominant hemodynamic disturbance in mitral valve disease.

Electrokymographic studies were made in 30 cases with rheumatic mitral lesions and the data were correlated with surgical findings in 28, with autopsy data in 2 A pattern of systolic plateau was found in all cases over the left atrium, and this was considered evidence of regurgitation However, while this plateau occurs early in cases with free regurgitation it occurs late in cases with predominant stenosis. These data allow a correct evaluation of the predominant hemodynamic disturbance

PANEL DISCUSSION ON LATE RESULTS OF MITRAL COMMISSUROTOMY

E COWLES ANDRUS (*Baltimore*), MODERATOR

SIR RUSSELL BROCK (*London*)
DWIGHT E HARKEN (*Boston*)
HARPER K HELLEMS (*Detroit*)
O HENRY JANTON (*Philadelphia*)
JOHN W KEYES (*Detroit*)
WILLIAM LIKOFF (*Philadelphia*)
ISIDRO PERIANES (*Buenos Aires*)
DOUGLAS ROBB (*Auckland*)

DR. ANDRUS

In conducting this panel it is proposed to direct questions to the members of the panel and to invite their comment on their experience. I want to open the discussion by asking, gentlemen, in your opinion what are the principal factors which influence the result of mitral valvotomy?

DR. KEYES

I think there are several major factors, but in our experience, complicating mitral insufficiency has had the most adverse effect on the mortality rate.

Certainly everyone knows how often we become suspicious preoperatively because of the presence of a systolic murmur, and at operation, there is not much in the way of regurgitation. I am going to quote our own figures on the presence of apical systolic murmur and how it alters mortality rate in the 153 cases we have studied so far

In pure mitral stenosis, we had 3 who succumbed out of a total of 94 If an apical systolic murmur was present, the figure was 9 deaths out of 33 So, although an apical systolic murmur does not invariably mean mitral insufficiency, it is one of the significant prognostic factors

SIR RUSSELL BROCK

This, of course, is a very difficult technical question. I think unless one talks for half an hour on it, one can simplify it only by saying that the first important thing is the correct diagnosis of mitral stenosis, in other words, the correct selection of the case.

The second thing that influences the operation is, what is done to the valve? That depends first on the condition of the valve—in other words, what the surgeon can achieve in view of the pathologic changes, second, what he can achieve technically If you have a favorable valve and a favorable surgeon, an experienced surgeon and a favorable selection, then you ought to get a good result.

surgery and in one by autopsy. The intermediate plateau was recorded in one case in all the projections and in 3 cases in several projections of the left atrium. Surgery confirmed a moderate amount of regurgitation in one case, some degree of regurgitation in another, while no regurgitant jet was felt in the third case. The late plateau appeared in one case in every projection, in 19 cases it was found in several projections; in the 2 others it was found in only one projection of the left atrium. Surgery confirmed some degree of regurgitation in 6 out of these 22 cases.

The results of our study raise several problems:

1. *Is the systolic plateau of the left atrial electrokymogram a true expression of mitral regurgitation?* There has been sufficient proof given by clinical and experimental studies of several groups in the medical literature, that the systolic expansion of the left atrial wall is a result of insufficiency of the mitral valve causing regurgitation of blood during ventricular systole. Pressure curves obtained in 1915 by Wiggers from the left atrium, in experimentally produced mitral insufficiency, demonstrate the same pattern. Electrokymographic and intracardiac pressure studies obtained in our laboratory in experimental animals show identical findings in dogs with surgically produced mitral insufficiency.

2. *What causes the delayed onset and end of the plateau in a predominant mitral stenosis?* In mitral stenosis, as opposed to pure mitral insufficiency, the diastolic pressure of the left atrium is high; therefore, regurgitation of blood will start only at that point at which the systolic pressure of the left ventricle reaches a height which is sufficient to overcome the atrial pressure, that is, during the phase of systolic ejection. After the opening of the mitral valve, the ventricular pressure decreases rapidly. However, mitral opening is delayed in comparison with cases without stenosis and the drop in pressure is slower on account of the narrowing of the mitral valve.

3. *What are the diagnostic advantages of serial electrokymography of the left atrium?* Delayed onset and delayed end of a plateau are therefore definite proof of mitral stenosis in the presence of some regurgitation, while an early plateau is evidence of free regurgitation without severe stenosis. A systematic study of the configuration of the plateau in the various projections, improvement of the calibrating methods, and correlation of all the graphic data may lead to a more precise determination of the amount of blood regurgitated, the condition of the left ventricle, and the localization of the lesion.

CONCLUSIONS AND SUMMARY

Electrokymography of the left atrium if applied correctly is the simplest and most reliable diagnostic method of recognizing which is the predominant hemodynamic disturbance in mitral valve disease.

Electrokymographic studies were made in 30 cases with rheumatic mitral lesions and the data were correlated with surgical findings in 28, with autopsy data in 2 A pattern of systolic plateau was found in all cases over the left atrium, and this was considered evidence of regurgitation However, while this plateau occurs early in cases with free regurgitation it occurs late in cases with predominant stenosis. These data allow a correct evaluation of the predominant hemodynamic disturbance

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DR. HARKEN

I think that is a fair summary of our position. We feel that if you get bad results from the surgical correction of mitral stenosis, it means that the selection of patients is poor, the surgery is poor, the postoperative care is poor or that there is a combination of all these things.

DR ANDRUS

Speaking as one of the internists present, I don't like to see all the blame laid on the internist

DR. HARKEN

The second and third factors both involve the surgeon. Are there other adverse factors beside the internist and the surgeon? I think that there are. I hoped something like this would come up.

Dr. Laurence B. Ellis has provided me with a list of seven adverse factors that influence the outcome. They are:

1. Age over forty
2. Atrial fibrillation
3. Associated aortic valve disease
4. Associated valvular insufficiency
5. Preoperative valve size of more than 1 sq. cm.
6. Postoperative valve size of less than 2.5 sq. cm.
7. Calcification of the valve

Any of those seven factors can be put into a formula and you arrive at a prognosis for the patient.

If none of these seven factors were present, the patients in our series had a 96 per cent chance of being improved If one factor was present (and there were 116 such patients), they had an 87 per cent chance of being improved; two, 72 per cent, three, 67 per cent; four, 56 per cent, five, 50 per cent There were 8 patients in this last category.

Of course, the prime factor controlled by the surgeon is the *quality* of valvuloplasty. He must understand the pathologic patterns possible. He must know that commissurotomy, i.e., simple separation of fused leaflets, is only part of a good operation He must open not only this primary or leaflet stenosis but the *secondary* stenosis as it is produced by fusion of the chordae tendineae and papillary muscles He must do all this without creating regurgitation or embolus.

Another point that must be made is the necessity for a prolonged (month) rigid medical regimen including an initial diuretic program. Somehow, our colleagues are constitutionally indisposed to such a program following surgery. They seem to feel that if the patient didn't need it before surgery and doesn't have peripheral edema, he doesn't need it once operation has been performed. This is just not so, and the poor old left ventricle is being asked to change suddenly from a protected resting state to an active vigorous state without a "training period."

In other words, I think there are factors that the internist, as well as the surgeon, can control. This will favorably influence surgery; and if results are consistently poor in a given clinic, one or more of these factors are at fault

DR. JANTON

I consider that the prominent factors in getting a good result are: first, that you have a valve that will accept an experienced surgeon's constructive surgery; second, that you have what Dr Harken has just beautifully presented —a real mitral valve block rather than myocardial insufficiency, third, that you do not have other valvular lesions

I think it is fairly important that we add right here that the eventual functional results of the best type of surgery depend upon whether or not the surgeon is operating in a potential bed of rheumatic activity, with the possibility of sequelae, and reactivation of rheumatic fever Hence, in ten years will we have a restenosis? These are the pertinent factors that I would consider

DR. HELLEMS

I think I can add very little In our group, we agree in general about the structure of the valve. My surgical colleague classifies these valves into three types the type that is freely movable, soft and pliable, which can be opened well, a type that is fibrotic, thick, and usually very difficult to cut, and finally, the type that is calcified, which constituted about 20 per cent of our group

He feels (and we agree with him on the basis of hemodynamics after this procedure) that even though in the fibrotic group and in the calcified group the surgeon opens the valve with his finger or with a knife back to the annulus, from a functional point of view and from a hemodynamic point of view this valve does not open properly under the pressures that are normally present in the left atrium.

Such a discrepancy does not mean that the surgeon is wrong in his observations, neither does it mean that the catheterization data are wrong after operation The surgeon tells us that he has opened the valve widely and that it will admit two or three fingers We do a postoperative hemodynamic measurement on that valve and we get an area that is much less than we expect to get. In other words, we have been disappointed, in our series at least, by the amount of change in valve size.

I am perfectly aware that these dynamic measurements at times are very difficult, because one cannot exclude changes in left ventricular filling pressure and so on, and they cannot be taken as perfectly definite. I think right heart failure affects this problem, as I am sure everyone knows The patient who has been in prolonged right heart failure repeatedly does not get the same result.

DR. ANDRUS

I don t want to prolong the discussion, but you speak as though you had absolute faith in the formula by which you arrive at the valve size. Do you think that was handed down from on high?

DR. HELLEMS

In our hands, for the first several cases (I can t give you the exact number), we made a very great effort to correlate the calculated valve size and the size of the valve as determined at operation, by drawings made with the surgeon,

and they agreed within 0.2 sq.cm., with the rarest of exceptions in which there were degrees of mitral insufficiency that we had not suspected. If they agreed before, and if we are dealing with a hemodynamic problem, I fail to see why they should not be adequate after the surgery if we are talking about a mechanical lesion and not a myocardial lesion

DR. LIKOFF
I think it is a plain and perhaps not curious fact that the internist in particular would like to ascribe failures in mitral commissurotomy to mechanical events, and in a certain respect perhaps that attitude is a just one, because the surgery of the mitral valve obviously depends upon many exquisite technical maneuvers, including a wide opening of the orifice and the correction of the subvalvular pathology.

However, there is another factor which I think has been expressed in a different fashion, concerning the ordinary evolutionary pattern of mitral stenosis and where in that evolutionary pattern the surgeon is asked to interfere. I think all of us will have to adopt the philosophy that it is impossible to ask of the surgeon a complete restitution of the valve and a reconstitution of the entire pathophysiologic process by means of a simple mechanical maneuver. If the patient has been allowed to deteriorate to a point where there is no normal return regardless of the excellency of the interference, then I think we are asking of him more than is humanly possible to accomplish.

Therefore, from an internist's standpoint I think it is important to recognize which of the groups of patients with mitral stenosis are bound to get away with their disease in its entirety and never come to difficulty (and I think that percentage is somewhere between 7 and 10 per cent of those afflicted), and which of the group has already embarked on a clinical toboggan from which there is no return, and the only question we cannot answer is the time element involved in that toboggan

I think the greatest fault or the greatest weakness in the entire approach to the problem is the selection of patients in their evolutionary pattern If you wait long enough, you will get the poorest results and the greatest list of complications.

DR. ANDRUS
You imply that the earlier they are selected, the better, or do you have other criteria that tell you where they are on the slide?

DR LIKOFF
The earlier we select them, the better, provided they are manifesting pathophysiologic events

MR. ROBB
What I have to say is very starkly clinical; and, like the other speakers, I feel that much depends on the state of the mitral valve and the roof of the atrium on the way to it, that is, excluding such considerations as the myocardial and the rheumatic complications that have to be differentiated

I would like to raise for discussion by this panel, for my own satisfaction if not for that of others, just how many mitral valves are in fact intractable (both the valve and the atrium) for access

Without having specifically added up figures, I would estimate that about 5 per cent of the atria do not permit access, and about 5 per cent of the valves, once one is there, are intractable by ordinary current means

Some of the difficulties of access are not insuperable, but when they are difficult they can add such complications as to make the success of the operation more doubtful and the risk of surgical complications greater

When one has reached the valve itself, one very frequently feels it very like a tight fibrosed anal sphincter, and one can hardly imagine being able to do much more with it even though one had it on a plate in front of him. That is really what I would like to ask the other members of the panel for—a rough estimate as to how many of these mechanical difficulties prevent a satisfactory result.

The assessment of these patients afterwards we have found to be notoriously difficult, if much reliance is placed on the story of the patient. Sometimes the most enthusiastic account comes from the patient in the category I have just mentioned, in whom one has made no impression on the valve.

Another point in the clinical aspect is the upgrading that one has been able to do between the immediate postoperative follow-up and the later one. Quite frequently the grades have improved over periods of six months or a year I think that is a useful point.

I would disagree profoundly with the speaker who said that advanced age is a contraindication. Some of our happiest results have been in patients who are near the 60-year mark.

DR. PERIANES

There are medical and surgical factors that have influenced the results of commissurotomy Since we do not feel able or qualified to discuss the former, we shall take up a discussion of the latter

We believe that the anatomic and functional status of the mitral valve is one of these important surgical factors The increase of the valvular motility can be expected in those patients in whom (1) the aortic leaflet of the stenotic mitral valve bulges toward the atrium during systole, (2) the size of the orifice varies during the cardiac cycle, and (3) there are slight anatomic changes

In these patients we have the higher percentages of objective improvement, including also the disappearance of the auscultatory signs of mitral stenosis in some cases Most of these patients belong to Group II or II–III of the A.H.A. classification.

We must not expect an increase of the valvular motility in those patients in whom (1) the aortic leaflet does not bulge, (2) the size of the stenotic mitral orifice suffers no variation, or very little, and (3) there are marked anatomic changes In these patients the mitral valve cannot move or moves very little after commissurotomy, and thus the functional result is poor Most of these

patients belong to Groups III and III–IV of the A.H A. classification. Other patients have characteristics of both groups.

The effect of commissurotomy on these patients' valves depends on whether they are closer to one or the other group These cases are the most frequent. This is the experience we can obtain with finger exploration of the valve.

We wish to emphasize the importance of a good functioning aortic leaflet to establish the late results of commissurotomy on the valve. All of these are general considerations useful as criteria when you deal with the individual case

DR ANDRUS

Mr. Robb has raised two points which I think we might lay before the members of the panel Do any of you wish to comment upon his question as to in what proportion of the cases the valve is in fact inaccessible?

DR HARKEN

I think there have been more than 900 operations since we last were unable to get to the mitral valve. Maybe we were unwise, and maybe the fine line between heroics and foolishness was crossed, but at least we did get to the mitral valve and haven't backed out since our first 100 cases

After we got to the valve I don't know how many we failed to help, but I suspect it is on the order of 5 per cent that would be regarded as morphologically hopeless.

This inability to get into the atrium includes patients who are being reoperated on and who therefore have no auricular appendage The primary operations were either in our hospital or elsewhere.

SIR RUSSELL BROCK

I wonder what happened to the first 100 cases.

DR HARKEN

I backed out three times

SIR RUSSELL BROCK

My series is very much smaller I have operated on only 350 with my own hands, but I have turned back only once, and that was in a fairly early case with, I thought, almost total thrombosis of the whole of the area of the atrial wall that I was looking at or feeling. I don't know whether today I would be unwise enough to go in, but otherwise I have not failed to reach the valve either at a first or a second operation

Concerning the percentage of cases in which you are unable to help the valve when you reach it, obviously that is going to vary in different groups Assuming that the condition is mitral stenosis and not mitral regurgitation, I don't know that I have ever had a case in which I could not help the patient to some extent, but of course there are some in whom the amount that you can do is very small. Some clinical results and indeed immediate hemodynamic

results are very good, although one may make very little increase in the size of the valve

DR. ANDRUS

Another point raised by Mr Robb was in disagreement with those who emphasize the adverse influence of age upon prospective benefit. Does anyone wish to comment on that?

DR. JANTON

I think Dr Harken mentioned the age factor of 40 as one of the seven variables that had to do with the functional results both now and in the future, rather than as a contraindication and one of the factors in getting a result.

Our oldest patient is 64 I would hate to have to report on a series of patients who were operated on at ages between 60 and 80 or 50 and 70

DR. LIKOFF

I too wish to reemphasize the fact that chronologic age is relatively unimportant as an indication or contraindication for commissurotomy I would like to use this as an excuse also for a further comment regarding the reliability of the subjective symptomatology of patients following mitral commissurotomy

Contrary to what has been stated, I have a deep respect for the subjective reports of the patients, for a very definite reason. These patients as a rule have been ill for a long period of time. Their disability is not recent, and they have an acute and an amazing appreciation of those things that they can do and with what sense of disability they can function. So, I think due credence must be given to their postoperative reports, provided a sufficient length of time has gone by for them to resume their normal activities

I think the statement that has been made, both in print and currently, that some patients are vastly improved when nothing has been done for them, is not at all surprising These patients who have been operated on, whose valves have been investigated, and who have been closed without any definitive procedure, are patients who have been treated with bed rest for a period of time. Following the unfortunate surgery they are put to bed as convalescent patients, and of course they feel better immediately They would feel better if nothing had been done. It is wise to go back into these histories and recheck the patients six months after they resume their activities You will find there is no such thing as psychologic relief from their so-called surgery, and they are truly just as ill as they were originally

DR. ANDRUS

Anticipating the shortage of time, I corresponded with the members of the panel and some questions were raised among ourselves Two of them have been hinted at but not completely covered

One question is in regard to the likelihood of improvement. It deals by implication with the likelihood of recurrence. The likelihood of recurrence,

it has been suggested by one member of the panel, should perhaps be influenced by many of the other factors including age.

Sir Russell, what has been your experience with recurrence of stenosis?

SIR RUSSELL BROCK

To date sufficient time has not passed since the greater number of valvotomies to estimate the final recurrence rate. In my own series of 350 I have reoperated on 4.

One of the basic troubles, of course, is the analysis of the state of the valve as found at operation, and what one can do for it. I think if one performs a complete valvotomy, opening both commissures completely, the chances of recurrence are very small. Obviously it must be a possibility, but there is little likelihood.

If you do less to the valve, then the chances of recurrence are greater; and certainly if you perform only a digital dilation of the central pathway, not separating the cusps, not separating the adherent chordae tendineae, then recurrence of stenosis sooner or later is inevitable.

I think it is really too early to give precise statistics.

DR PERIANES

I agree with Sir Russell. I might add that we can have recurrence even if we do a correct commissurotomy, if we have not very much motility

In 18 cases we have examined, there have been 2 cases of recurrence.

DR. ANDRUS

What effect does persistent rheumatic disease have on the likelihood of recurrence?

DR. HELLEMS

Regarding this problem of recurrence, in our series of 26 patients who were operated on before July 1953, and who have now gone two years after the operation, we had 3 patients with apparent reclosure of the valves.

One patient had a pliable valve and both sides were opened at the commissures, by knife and finger. His valve size before the operation was 0.8 sq cm. It went to about 2 sq.cm. two months after surgery He had an episode of active rheumatic fever eight months postoperatively. We restudied him after he was completely compensated, in about eighteen months, and his valve size was then smaller than it had been before. (I am using the valve size as an expression of changes in pressure and changes in flow.) I think as near as one can tell, this patient did have an open valve, and then had resealing

The other cases are not so clear-cut. They had calcified valves In each case at post-mortem the valve was like a rock, and it was hard to tell what if anything had been done. I was not sure they had been opened and no one else was sure they had opened. Recurrence is not a practical problem in recommending the procedure I think, however, that we will see recurrence in something like 4 to 5 per cent of the patients.

DR. KEYES

We have reoperated on one case out of our 180 There are 4 more that are clinically suspected of recurrence. One of these 4 definitely had a recurrence of rheumatic fever manifested by typical migratory arthralgia and fever During that period she probably had the so-called commissurotomy syndrome.

DR. ANDRUS

Then there seem to be two possibilities—first, what in the present company we would say is unlikely, that the valve had not been adequately opened, and, second, that either continuous rheumatic disease in the myocardium leads to reclosure or intercurrent recrudescence of rheumatic disease in the heart leads to reclosure by progress of the process which originally caused the stenosis Is that your idea?

DR. KEYES

Yes I think they closed once with rheumatic fever, and if the patient continues to have it they will probably close again.

DR. LIKOFF

We have had about the same experience that everyone else has had as far as numbers are concerned, having reoperated on 4 cases for so-called closure out of approximately the first 1000 cases that were done.

However, I want to call your attention to another possibility, namely, the reexamination of the symptoms that indicate a so-called reclosure.

I think a certain number of patients who manifest right-sided heart failure following mitral commissurotomy after some time may truly be suffering from tricuspid stenosis that had not been diagnosed at the time the mitral commissurotomy was done.

In several instances we have found that to be the explanation, rather than the reclosure of the mitral valve. Probably somewhere around 5 per cent of the cases with mitral stenosis have attendant tricuspid stenosis, and we have a great deal of difficulty in being able to diagnose it clinically

DR. ANDRUS

Do you think that what is mistaken for reclosure on the basis of dynamics may be based on irreversible pulmonary hypertension?

DR. LIKOFF

Yes, I certainly would echo that thought.

SIR RUSSELL BROCK

It is very important to realize what the last speaker said about this confusion between restenosis and clinical deterioration. It does not follow that because the patient's condition has regressed, it is due to stenosis

One of my colleagues recently analyzed 50 cases that had been operated on at least three years before, and the facts he emerged with were that there were factors such as the onset of atrial fibrillation, the persistence of hyper-

tension, the presence of emphysema and chronic bronchitis, and persiste of the degree of regurgitation either at the time of operation or caused by operation Those were just as important as the possibility of restenosis.

It has been our experience that the recurrence of rheumatic fever, althoı the patient may be quite ill at the time, has not necessarily been associa with any impairment of the final result Although in theory, of course, valve cusps (if they have once fused) could fuse again and indeed may f again, I think we must not ignore the fact that the valves as they were oı inally were primarily healthy and presumably had some vascularity. H(ever, after the valvotomy we are dealing with a valve which may be qı different anatomically and pathophysiologically In other words, it may avascular and it may not be subject to the same laws of fusion.

DR HARKEN

I think we have passed a little too lightly over the fact that the surge may not have in fact fixed the mitral stenosis. In the 10 cases redone out the 800 I have available for study, we can say that I predicted an unsat factory result in 9 So, in only one did I think at the time of the operatior would be satisfactory and drew a diagram of a satisfactory operation.

DR. ANDRUS

Were you using the knife or the bare finger at that time?

DR HARKEN

All those that have been reoperated on were done before I began to use t valvulotome satisfactorily.

DR HELLEMS

I would like to raise a question regarding this problem. I think we oug to define what we mean by an adequate opening of the valve If we me physiologic normality, complete hemodynamic normality, then we have n experienced such a situation We have yet to operate on a patient who, c both rest and exercise after the operation, shows a completely normal hem(dynamic pattern. I am confident that the one case I described had a recuı rence of stenosis in the incised valve.

DR. ANDRUS

That point is worth emphasis—that however wide the valve may be openec this condition has been present for a long time, has worked other alteration in the circulatory system which, leaving out of consideration the possibilit of further deterioration by continuing disease, we cannot expect to correc by simple interference at the point of the dam.

The questions that have come from the audience have included a natura projection of what we have just been discussing, namely, this:

Assuming that continuous rheumatic disease in the myocardium at variou levels, or recurrent rheumatic fever following streptococcus infection, may

play a part in the adverse effects which follow commissurotomy, what can be done about it?

Specifically, in the opinion of any or all of the members of the panel, is it likely that prophylaxis with sulfadiazine or penicillin can favorably influence the subsequent course of these individuals?

DR. JANTON

I certainly believe that the prophylaxis of giving penicillin before and after surgery does a great deal toward reducing the postoperative incidence of subclinical rheumatic fever which has been present, but which we have not been able to diagnose.

We know that even under fairly high dosages of penicillin these patients can have a flare-up of rheumatic fever which may not be due to the surgery That is, the patient may have pleuritis and pericarditis that he did not have one year previous to operation This is something new, above and beyond traumatic pericarditis, above and beyond pain in the chest due to retained fluid, and therefore we feel that if the patient has pleuropericarditis as a syndrome occurring six weeks after surgery, it is cyclic in nature. In our hands this occurs in between 20 and 30 per cent of our patients

I would hesitate (because I have not done a detailed analysis) to say how many of my patients, who in many instances live far away, stay on penicillin prophylaxis I do know that when they return for their six-month check-up it is rare to find a patient who has been feeling good It is that group of patients who not infrequently *will* flare up in the six-month *interval* after they leave us, and they have made up perhaps our biggest bulk of those with the so-called pleuropericardial syndrome.

One other factor It is well known that we do not know much about rheumatic fever We have no specific tests for it. We are up against something called rheumatic fever, and the best approach we have had has been prophylactic penicillin. Recently we have reported several deaths in patients above the age of 50 who had evidence of definite rheumatic myocarditis So, acute rheumatic fever *does* occur in the older age group, and probably the percentage will be greater as our diagnostic acumen becomes greater

DR. LIKOFF

It is our custom routinely to continue prophylactic antibiotic penicillin or sulfadiazine postoperatively for approximately six months, to circumvent what is described as acute rheumatic activity

I think one of the most important features of this whole matter is the definition of so-called acute rheumatic activity and what we are trying to prevent. Unfortunately, at the present time all of us use a loose terminology and, as a result, our thinking in the matter is quite confused.

In our own limited manner we have attempted to define reactivated rheumatic infection and have failed, as have several others We have failed in the respect that we cannot utilize to advantage any of the common diagnostic measures indicating rheumatic activity in these patients Perhaps this is not strange, in view of the ordinary clinical background of rheumatic activity in

adults As we all know, it is not common, and it is only of sporadic occurrence in other types of surgical intervention in patients who have had past rheumatic activity.

Nevertheless, our serologic studies, in a very carefully guarded group, revealed that there was no response in positivity in those who had this so-called reactivation as opposed to those who did not The statistical analysis was quite similar in 100 cases that were watched carefully for one year.

Despite that fact, however—and I am pointing it out to illustrate our own inadequacies in the matter—we continue to give the antibiotic as an anti-streptococcal medication after surgery, and we do so, we believe, with some reasonable change in the comfort of the patient as far as local respiratory infections are concerned afterwards

DR ANDRUS

Do you do it for six months?

DR LIKOFF

We do it for six months

DR. ANDRUS

If I may comment here, I confess to the same inability to identify rheumatic fever by serologic examination. What one is testing for is evidence of recent streptococcus infection in more instances than not. These individuals may either get a streptococcus infection and by implication a recrudescence, or they may have a process which is continually smoldering and which in the end may determine the outcome of their disease We lack tests to estimate the severity of that, and in all probability we still lack methods of controlling it medically.

The matter of the postoperative commissurotomy syndrome has been raised. Does anyone wish to comment on that? Is it recrudescent rheumatic fever? Is it something entirely different?

DR. LIKOFF

Instead of just rehashing all the old material that you all know about—that there is a syndrome which develops after operation, which is cyclic, phasic, peculiar and apparently related (at least we believe it is) more to the collection of pleuropericardial fluid that has some old blood in it than anything else, there is this interesting fact

Apparently our department of pathology has been able to isolate, by staining technique, several different types of Aschoff nodule bodies. There is that body which manifests itself by certain staining techniques and apparently is typically chronic, and there are two other types of bodies that represent subacute and more acute rheumatic activity.

The interesting part of the concept is simply that apparently there is no correlation so far of the more acute forms of the Aschoff body, according to these staining techniques, and the recrudescence of fever postoperatively. That is not definitive, I realize, but it may be taken for what it is worth.

SIR RUSSELL BROCK

This is a subject that has been gone over and over almost ad nauseam, but I would like to make a couple of comments

The first thing is the acceptance that this condition, whatever it is, is related only to operations upon the rheumatic heart. I think there is little doubt that throughout the world the main experience of most cardiac surgeons is in operations upon postrheumatic conditions

That is exemplified in the very small numbers, for example, of direct operations on the heart in congenital heart disease. I myself have done about 250 direct operations on the heart in congenital heart disease, and I suppose I have opened the pericardium and explored the heart without necessarily doing an operation on it in about the same number of cases

I think that is an experience which probably is greater than that of quite a number of people whose chief experience has been in postrheumatic cases There is no doubt at all that you get a considerable incidence of this so-called postcommissurotomy syndrome when operating on the heart in congenital heart disease in the absence of rheumatic fever, not in addition to it. I have operated on patients with congenital heart disease who have had active rheumatic pericarditis at the time of operation, and they have not had this syndrome afterwards

DR. KEYES

Just one comment. We have been interested in this, also

I don t believe our incidence is as *high as has been reported It is probably* less than 15 per cent in our group However, we have had some very severe cases One case has had a prolonged P-R interval and atrioventricular block, associated with pericarditis We have not been able to correlate the antistreptolysin titer elevation or the clinical and laboratory evidence for rheumatic fever I do think it is well known that chronic, smoldering rheumatic fever can continue to exist, and whether or not it can be reactivated as a result of stress of surgery is a question.

DR. ANDRUS

I should like to say something apropos of Sir Russell's remarks Before leaving Baltimore, I put this question up to Dr Blalock. We had 18 cases of this syndrome in the first 150 commissurotomies His reply was that in his experience the incidence of such a condition after operations, even those involving the heart, principally for congenital defects, was nowhere near that high. He did see it, but it was rare.

DR. PERIANES

We feel that in the so-called postcommissurotomy syndrome, rheumatic fever does not have very much influence. We believe it is an operative condition. We have had a higher percentage of the so-called postcommissurotomy syndrome in those cases in which we approached the heart through the anterior rather than the posterolateral wall

DR. ANDRUS

Sir Russell, in your letter to me, you raised one question that you might briefly touch on. Having in mind the possibility of recurrence, does the age of the patient influence your selection?

SIR RUSSELL BROCK

This is an important matter, particularly in regard to the question of operating on patients very early in their illness

I think Dr. Likoff mentioned this matter, and I agree very strongly with what he said—that provided the patient is beginning to get symptoms or features of disturbance as a result of his mitral stenosis, then the earlier he is operated on, the better. On the other hand, I think one must take into account this question of advising operation on a patient in his twenties. I have operated on many patients in their twenties, on whom one is compelled to operate because of the severe secondary mechanical effects.

Although I think that restenosis (particularly after an adequate operation) is uncommon, one must accept the fact that in a patient who is going to live for another thirty, forty or fifty years, the possibility of restenosis must come up, and therefore one must pay attention to that in considering the question of operation on a young patient

I don't want to be misunderstood. I like to operate on patients early, and if they have mitral stenosis it ought to be relieved On the other hand, I think there must be a certain amount of restraint in very young patients

DR ANDRUS

We are bound to omit some of the most vexing and contentious questions on this whole subject, but one of great importance is the matter of the significance of embolism in the selection of patients, and the means of controlling (if possible) embolization during operation. Does anyone wish to comment on that?

DR. HARKEN

I can say that rather than representing a contraindication to operation, frequent embolization before surgery, in our opinion, is a firm indication for surgery, and the fact that in the first 500 patients referred to earlier, with a thirty-month follow-up, there have been but 5 emboli, seems to support the contention that removal of the appendage and change of the hemodynamics of the left atrium will probably reduce the incidence of embolus.

DR ANDRUS

There have been only 5 instances of embolus after operation, during the subsequent course?

DR HARKEN

Yes. We are taking out of that group the patients who have emboli produced by surgery.

DR. ANDRUS

Do you want to discuss that problem?

DR. HARKEN

I would like not to, but I will do it I would rather discuss it in the last 300 patients

We have had 11 per cent of emboli in the Group IV patients, and 2 6 per cent in the Group III patients It was twice that high in the first 100 patients In other words, we are down to less than 3 per cent in Group III and around 10 plus per cent in Group IV

How have we influenced that? With standard maneuvers, which most of you use, such as flushing the atrium. Head vessel isolation is now used only with calcific valves We are a great deal more careful in the manipulation of the valve than we used to be.

SIR RUSSELL BROCK

There is no question, of course, that the incidence of embolism after satisfactory mitral valvotomy is very small In fact, I have not had a single case of embolism occurring after operation—again not counting those that were precipitated by operation.

The thing that worries me is the policy of routine control of the head vessels I rather gather from what Dr Harken just said that he does not now do that as a routine. If that is so, I am glad to hear it, because, although I use this maneuver frequently, I think the other things he has mentioned are of equal importance, namely, flushing the atrium before one handles it, handling the valve very gently, and of course selecting cases in which one should control the head vessels

The thing that worries me is that by controlling the head vessels you can actually precipitate the very condition you wish to avoid.

MR. ROBB

I, too, am glad to hear this discussion, not having had a very clear policy in our unit as to what to do about this In fact, we have done very little in the way of controlling the head vessels, and I am glad to have this confirmed

DR. KEYES

In regard to embolic deaths, we have had a total of 3 in our series Two of them occurred in the immediate postoperative period. However, we did have a saddle embolus that occurred during the immediate postoperative period, which was successfully removed, followed by a second late recurrent saddle embolus, also successfully removed, and the patient is now living and doing very well.

DR. ANDRUS

May I address a question to Dr Harken and Sir Russell in regard to their recent remarks

Given a patient who has no serious circulatory disability with mitral stenosis, and who has had an embolism, would you regard that as an indication for operation?

DR. HARKEN

We have not operated on such a patient.

SIR RUSSELL BROCK

I don't think it is quite as easy as that. There usually are some factors that are helpful. I presume that it is almost certain that this patient is in a state of atrial fibrillation, or has had episodes of it. If the patient were in atrial fibrillation and had an embolism, I would advise operation, although I think we have all found in those cases that there may be no clot at operation. The disasters that may befall the patient are so great that I am never happy leaving a patient who has had one episode.

DR. ANDRUS

There is a recurrent question also, namely, whether amputation of the auricular appendage is necessary, or whether it is sufficient to do that alone. You would always, I take it, do a valvotomy if you got that far?

SIR RUSSELL BROCK

Oh, yes.

DR. HELLEMS

I would like to agree with Sir Russell somewhat. We have had 10 patients operated on for emboli alone. In other words, they were practically asymptomatic, but they did have episodes of emboli.

We consider it an indication regardless of the underlying symptoms, because we feel that even though the patient has not had many symptoms at that time from a hemodynamic point of view, it still indicates a stasis phenomenon in the left atrium, and when the surgeon has operated, the valve has been found to be quite small.

We had two cases in the early days in which we just removed the auricular appendage and did not relieve the stenosis. They were both Grade IV patients, and they both had recurrences of the emboli, and both died of it, because the stenosis was not cured.

DR LIKOFF

Just a word of caution about the clinical picture of emboli

Emboli are not isolated incidents in the course of mitral stenosis If you trace back the history of these patients over a period of ten years, you will find that 60 per cent of them had two emboli within a seven-year period of time, and a little over 75 per cent of the patients will have two emboli by the time a decade has rolled by.

So, I do not think it is truly fair to look at this as an isolated incident that will never recur It is potential trouble for the future, although it is no

more and no less potential trouble than the disease itself, and it has to be approached with the same philosophy

Therefore, I would echo very strongly what has been said. An embolic episode is a primary indication for mitral commissurotomy

DR. ANDRUS

There is a question from the audience which I will take the liberty of answering myself

Is it possible to anticipate which case will have clots or emboli?

In reply, I think one should say that patients with atrial fibrillation, those with evidence of activity, and those with a more severe degree of circulatory failure, are those in whom embolism is most likely to be anticipated. At operation, even with sinus rhythm and in the absence of these other phenomena, of course, a calcified valve presents a particular hazard

So far in this discussion nobody has mentioned Dicumarol Is that of significance, or is it regarded just as a form of holy water that is good for the soul of the person who gives it?

DR. JANTON

Quite a few of our patients have come to us who have been on Dicumarol over a period of one, two or more years The interesting thing is that at operation in every one of these patients there was a clot either in the auricular appendage or the left atrium, and right in the center the clot was soft and of currant jelly consistency That always presents a hazard. Whereas the nice, very fibrotic clot can be dissected without the production of an embolus, these people who have been on Dicumarol for any length of time present a problem.

DR. KEYES

We have been using anticoagulants rather extensively for peripheral emboli in rheumatic fever Our figures are very small and it is very difficult to state whether or not they are of value.

Personally, I feel that several patients I have observed, who have had multiple emboli, have been improved after using Dicumarol. In a patient who has been operated on and who has continued to show emboli, I think you have nothing else to offer him

DR. ANDRUS

At what level do you recommend keeping the prothrombin time?

DR. KEYES

Twice normal control time in seconds is a reasonable level.

DR. HARKEN

This clinical syndrome of a person who has been operated on and who continues to throw emboli is rare

In the third, fourth and fifth hundreds of our original 500 patients, those

who had emboli in Group III were those who were past 40, who had calcific valves, who were fibrillators, or those who had thrown emboli in the past.

DR. ANDRUS

In the first 150 patients at Hopkins, we had 5 patients who had embolism subsequent to operation. Two of those were interesting, in that they had it while they were still in the hospital, about ten days after operation. They were almost ambulatory. We inferred, perhaps mistakenly, that it might be due to some clot formation along the suture line. This has been demonstrated in patients who have died ten to fourteen days after operation, and in that little cul-de-sac against the sutures there is a small thrombus.

DR. HARKEN

But in that era I think we were leaving a lot more appendage than we leave now.

DR. LIKOFF

In the group of patients with emboli that we reviewed, the effect of Dicumarol was studied Slightly over 25 per cent had experienced embolic issue, and that statistic remained constant when they were without Dicumarol. The percentage of recurring emboli dropped to 13 per cent when the patients were carried on Dicumarol. So, apparently there is some justification, in those who are not operated on, to continue with that form of therapy.

The incidence dropped even further after operation—below 5 per cent. From a technical standpoint, apparently, it is wise that once a patient has been placed on Dicumarol, he should be kept on the drug if he is coming to surgery, until immediately beforehand when it is withdrawn, and perhaps right up to the point of operation the patient's blood coagulability can be controlled with quicker-acting drugs such as heparin.

THE TECHNIQUE OF MITRAL COMMISSUROTOMY

ROBERT P GLOVER (*Philadelphia*)

The method of mitral commissurotomy which I will describe is one which I have used over the past six and one-half years This method with little variation has been employed in a series of 750 cases by me and, with some minor modifications, by my associate, Dr Thomas J E. O'Neill.

PREOPERATIVE MEDICATION AND ANESTHESIA

Most anesthetists are agreed that regardless of the particular drugs used for anesthesia, patients undergoing intracardiac surgery tolerate large doses of anesthetic agents poorly The entire cardiovascular system, central and peripheral, may be rather markedly depressed by what in the average patient would amount to a moderate or small amount of anesthesia.

At the Presbyterian Hospital in Philadelphia, Dr Seymour Schotz and his associates have worked out an anesthetic technique which is well tolerated by cardiac patients Very little preanesthetic medication is given. The patient receives Seconal gr ¾, by mouth, and atropine gr 1/150, by hypodermic, one hour before surgery Anesthesia is induced with Pentothal sodium and relaxation for intubation obtained with succinylcholine. Anesthesia is maintained with 50 per cent nitrous oxide and oxygen. Enough succinylcholine by intermittent intravenous drip is added to control the patient's muscular activity as required for the surgery After induction, before the pleura is entered and after it is closed, the succinylcholine is not needed. While the pleura is open and especially during the actual cardiac operation, active respiratory activity on the part of the patient is abolished by the intermittent administration of the succinylcholine, efficient pulmonary ventilation being maintained by inflation of the lungs using manual bag pressure. About 20 to 30 minutes after the Pentothal is discontinued, most of it is eliminated and the patient is carried along on analgesic mixtures of nitrous oxide and oxygen. Patients will often respond to commands by opening and closing their eyes and by nodding or shaking the head. Apparently the nitrous oxide analgesia is sufficient to provide not only a reasonably good analgesia but also a complete amnesia for the period of operation. On occasion when the nitrous oxide has been discontinued or its flow markedly diminished, patients have reported having overheard some comment made in the operating room. This can be avoided by maintaining a 50 per cent level of nitrous oxide in the inhaled mixture.

Observations over the past two years have led to the following conclusions

(1) This combination of nitrous oxide analgesia and succinylcholine for the control of muscular activity provides a type of anesthesia which is easily controlled, does not appear to tax an already compromised myocardium and appears to produce few, if any, undesirable side effects. (2) In spite of the very light levels of anesthesia (first stage Guedel), the incidence of various arrhythmias is no higher than under more conventional methods of anesthesia.

It has not been necessary to use procaine intravenously to depress cardiac irritability as a routine. It is used in a 0.2 per cent concentration intravenously only if the patient shows evidence of increased irritability by the presence of more than an occasional ectopic beat. Since all patients are monitored by using an oscilloscope plugged into a direct-writing electrocardiograph, it is felt that these arrhythmias can be detected early. The only other patients who receive intravenous procaine or any other drug designed to diminish cardiac irritability are those who come to the operating room with a tachycardia and in whom the tachycardia does not subside as they go to sleep. In the last 200 cardiac operations at the Presbyterian Hospital in Philadelphia only one serious cardiac arrhythmia has been seen which could not be explained on the basis of a surgically induced period of coronary ischemia. There is great virtue in using as few drugs as possible so that one is not confronted by a serious circulatory situation which cannot be easily diagnosed.

Early in this program there was a tendency to be too sparing with the use of blood transfusions. A number of serious hypotensions resulted. Since the practice of administering blood to all patients with the start of surgery has been adopted, much less difficulty has been noted and no patients have been thrown into pulmonary edema by overloading. The usual patient undergoing mitral commissurotomy will receive 500 cc. of whole blood. Should there be more than the usual amount of bleeding, one or more additional pints is, of course, administered.

On occasion a serious hypotension which could not be explained on the basis of blood loss has been seen. These patients have responded well to vasopressor therapy. A dilute solution of methoxamine (20 mg to 500 cc) is started and if this is not effective, a change to a drip of norepinephrine (4 mg. to 1000 cc) is used.

One of the advantages of the anesthetic technique described is that the patients are fully awake when the nitrous oxide is discontinued. One can then determine whether peripheral or cerebral emboli have occurred In the former, embolectomy is performed. In the latter, immediate stellate ganglion block is done. Seventy per cent of the patients so blocked have responded well although one cannot be sure that they might not have responded well without this therapy.

Under such a regimen, which includes frequent tracheobronchial aspirations as indicated, it is rarely necessary to perform bronchoscopy at any time during the postoperative period.

THE INCISION

The patient is placed in the right lateral decubitus position. A standard left posterolateral periscapular incision is used The advantages of a postero-

lateral approach are many The left atrium is posteriorly placed, its exposure by this approach is complete and readily accessible should complications such as a thrombosed appendage or hemorrhage be encountered. Even more important, proper valvular manipulation and reconstruction is unhampered for the intra-atrial finger and knife is in a more proper and mobile plane. The posterolateral incision is carried from the tip of the scapula posteriorly, parabolically forward to the midclavicular line, its anterior extension lying in the submammary crease. Very adequate exposure can be obtained by entering the pleura through the fourth interspace, it not being necessary in most instances to remove a rib The lung is retracted posteriorly to expose the heart lying in its intact pericardium. The lung itself will usually be somewhat turgid, thickened and even rubbery in consistency but is expansile.



Fig 1 Pericardium partially opened vertically posterior and parallel to the left phrenic nerve. Lung retracted posteriorly patient in true lateral position with the left side up head to the right and legs to the left.

INTRAPERICARDIAL APPROACH

The pericardium is opened vertically *posterior* and parallel to the phrenic nerve (Fig. 1), carried superiorly to above the level of the pulmonary artery and inferiorly to a point midway between the apex of the left ventricle and the circumflex coronary artery at the mitral annulus The anterior flap of pericardium with its contained phrenic nerve is then reflected forward to be loosely attached to the anterior chest wall for complete exposure Opening the pericardium posterior to the phrenic nerve is advantageous for it gives a more complete exposure to the entire auricular appendage, the left atrium and the pulmonary veins (Fig 2) Although exposure of this completeness may not be necessary in the average mitral commissurotomy, it is of paramount

importance should the appendage be small and contracted, should direct access to the left atrial wall be necessary or should hemorrhage ensue from a split appendage or atrium during valvular manipulations.

At this point complete exploration of the exposed portions of the heart should be made. An estimation of the size of the cardiac chambers, pulmonary artery and pulmonary veins is made and recorded. Palpation of the left ventricle at the apex will reveal the typical diastolic thrill of mitral stenosis. Similar palpation over the entire left atrium will reveal the presence of a systolic thrill indicative of a significant degree of associated mitral insufficiency. The additional presence of aortic valve disease should always be sought Although the aortic valve cannot be palpated directly, a great deal



Fig 2 The necessary exposure of the heart for proper commissurotomy Anterior flap of pericardium sutured to anterior chest wall, the left ventricle to the left, the pulmonary artery anteriorly and superiorly, the left auricular appendage protruding from the well exposed left atrium and the pulmonary veins in the foreground

can be learned by palpation of the ascending aorta and the aortic ring This is accomplished by sliding the thumb of the left hand into the transverse sinus beneath the pulmonary artery over to the posterior surface of the aorta, and the index finger anterior to the pulmonary artery on to the anterior surface of the aorta (Fig 3). In this manner the entire ascending aorta can be palpated, and the presence of a telltale systolic thrill and poststenotic dilatation will be direct evidence of the presence of aortic stenosis By snugging these two fingers down to and even onto the myocardium at the root of the aorta, the actual aortic ring can be palpated and squeezed

Should appreciable aortic stenosis be present, thickness and induration of the aortic ring and surrounding myocardium and even the presence of calcium can be readily detected. Should aortic valve disease be suspected by

this maneuver, direct pressure tracings are taken in the left ventricle and the aorta. A gradient of more than 25 mm. Hg from left ventricle to aorta is usually indicative of appreciable aortic stenosis If the gradient is low (25 to 50 mm. Hg), one will proceed to the originally planned commissurotomy, but at its completion left ventricular and aortic pressures must be repeated. An appreciable increase in the gradient or the development of significant mitral insufficiency demands relief of the aortic valve obstruction

Should, of course, a very marked aortic stenosis be present with a high gradient, it may be advisable to proceed with aortic commissurotomy before mitral commissurotomy, for physiologically speaking the aortic stenosis should be relieved before the mitral stenosis Early in our experience, primarily for

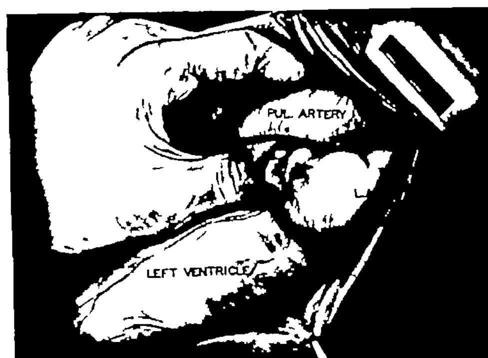


Fig 3 The maneuver for external palpation of the ascending aorta and aortic ring. The left thumb is introduced into the transverse sinus under the pulmonary artery and the index finger placed anterior to the pulmonary artery onto the aorta

technical reasons, we frequently resorted to mitral commissurotomy prior to aortic commissurotomy, and this may be well tolerated provided the aortic valve is opened promptly thereafter Our original thinking was dictated by the fact that mitral commissurotomy is the much easier procedure not only to perform but for the patient to tolerate. Thus, because of the trauma to the left ventricle, it seemed advisable to leave that portion of the combined operation to the last. In more recent cases, however, with improved instruments and technique for aortic commissurotomy through the left ventricular myocardium, we proceed to the aortic valve before the mitral valve. It might be wise, however, to place the purse-string suture around the base of the left auricular appendage before proceeding with the aortic commissurotomy so that little time will be wasted in gaining access to the mitral valve.

Having completed the proper external exploration of the various cardiac

chambers, one will then proceed directly to the performance of miti missurotomy.

MANAGEMENT OF THE LEFT AURICULAR APPENDAG

Prior to any appreciable manipulation of the left auricular app gentle palpation of this structure for evidence of contained thrombos be made. Should thickening and induration be present in the pres atrial fibrillation, it is likely that thrombosis has taken place within pendage and greater care is then taken as one places the purse-strin; its base. There are those who prefer at this point to place a clamp o base of the appendage before placing the purse-string, feeling that

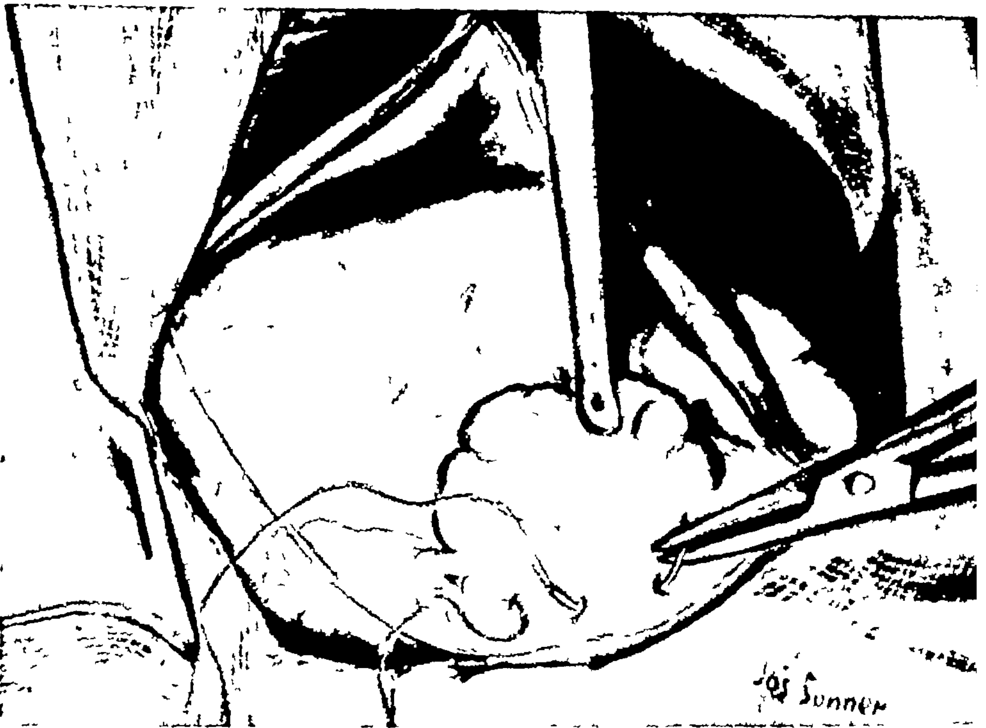


Fig. 4 Purse-stringing the wall of the left atrium just proximal to the l the left auricular appendage The suture begins at the inferior angle of the app just above the circumflex coronary artery

manner emboli are less likely to be produced. This is debatable and we ; to place the purse-string suture first. Care, of course, must be taken i manipulate the appendage more than is absolutely necessary.

Although it is frequently stated that the left auricular appendage is p stringed, actually in our experience the purse-string suture is placed *pro to the base of the appendage* on the atrium proper (Fig 4) This has se advantages. (1) The atrial wall itself, as a rule, will be a sturdier stru and less likely to tear. (2) The purse-string suture is applied much proximally to any contained thrombotic material making the producti emboli less likely. (3) At the conclusion of the procedure when the p string is tied, the size of the atrium proper will be somewhat reduced presumably may therefore have a better ejection force (4) By more adequ reducing the size of the left atrium at this lower level, should any tend

to jet-like regurgitation through the anterolateral commissurotomy near the myocardium be present, a purse-string so placed will pucker the tissues in this region buffering the left atrium and minimizing the regurgitant effect.

Placement of the purse-string is begun by taking a small bite at the inferior angle of the protruding left auricular appendage just above the circumflex coronary artery Small bites are taken in the myocardium completely surrounding the base of the appendage, beginning on the lateral surface and extending superiorly into the transverse sinus (Fig 4) It is well to take one or two fairly sturdy bites below the superior aspect of the appendage, for in this area there is usually localized bulging and thinning of the myocardium

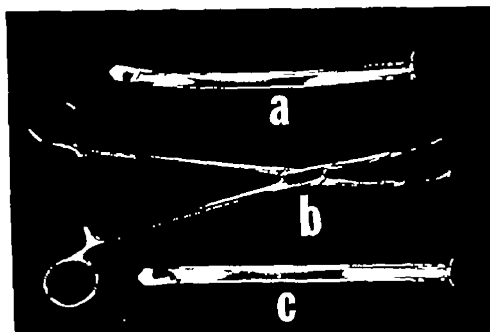


Fig 5 Specialized instruments for mitral commissurotomy *a* and *c*, The left and right commissurotomy guillotines respectively (made by Bruno Richter and Company) Note the triangular-shaped hook designed to be introduced through a mitral orifice of any size. This hook is completely dull and is used to engage and immobilize the valve commissure, not to cut. The sharp but protected guillotine blade does all the cutting from the atrial side of the valve to avoid injury to chordae tendineae. *b* Auricular appendage clamp with serrated blades designed to grasp the atrial wall without slipping (made by George Pilling and Company)

and this may well be the weakest portion of the purse-stringed area. The purse-string is then carried medially and downward, parallel to the circumflex coronary artery to join its initial point of entrance. The two ends of the purse-string may be placed in a Rumel tourniquet if considerable exploration and manipulation is contemplated although we prefer, in the routine mitral commissurotomy, to control the purse-string manually The first turn of a square knot is placed and the two ends left long so that the assistant can tighten or loosen the ligature as desired. The neck of the appendage *in one* half of the cases will be small, fitting snugly around the intracardiac finger so that very little, if any, tension need be placed on the purse-string itself. In the others very little tension is necessary so that mechanical tourniquets are usually superfluous

A properly appointed clamp equipped with a serrated grip which will not slip from the smooth, rounded atrial myocardium is applied to the atrial wall proximal to the base of the appendage (Fig 5*b*). The employment of such a clamp, so placed, excludes all thrombotic material from the circulation except in those instances, fortunately uncommon, where thrombosis is widespread throughout the left atrium. The tip of the excluded appendage is then opened but not amputated (Fig. 6). It is preferable to leave all appendage tissue attached at this stage of the operation for later use as a pedicle graft should undue bleeding occur from the atrium or ventricle. This, of course, is rarely necessary but on occasion may be highly desirable. The incision in the appendage is made just large enough to easily accommodate the right index

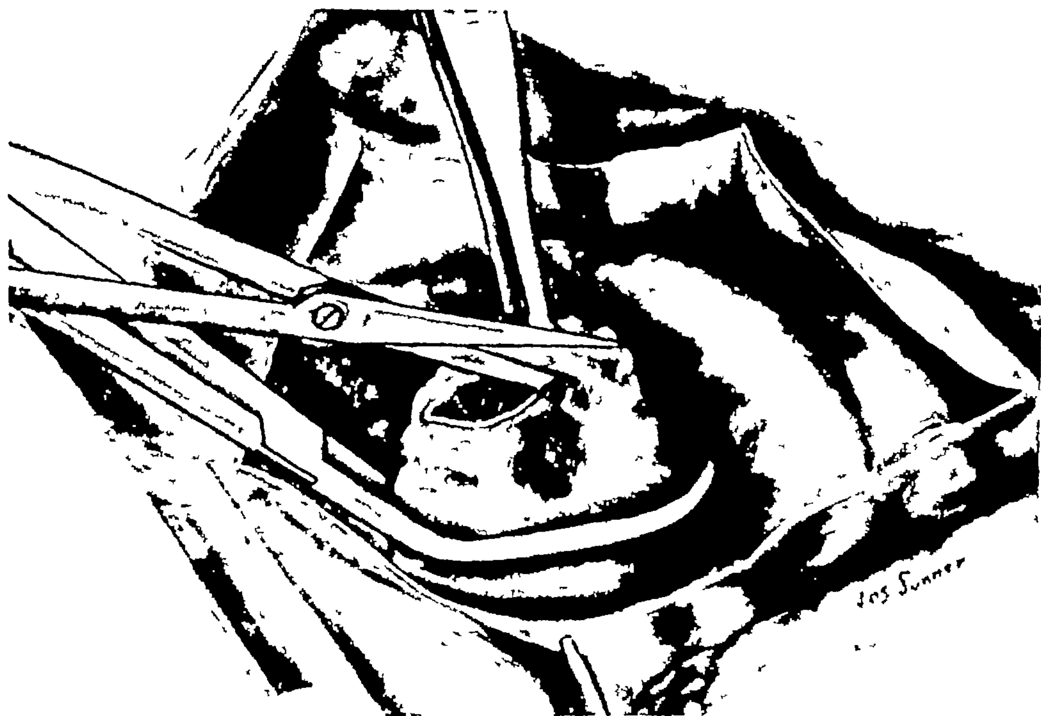


Fig. 6 The tip of the auricular appendage opened just below its tip No amputation of the appendage is made until the commissurotomy is completed

finger. The lumen of the appendage is explored for evidence of thrombotic material Whether such material is seen or not, the interior of the appendage is flushed repeatedly with saline solution (Fig. 7). When clean, the clamp is loosened permitting a controlled gush of blood to surge from the left atrium outward through the appendage, washing out any particles of thrombotic material which may be present at the site of the clamp. This may be done two or three times if necessary (Fig 8) The appendage is now prepared to receive the right index finger.

The finger is inserted with its palmar surface toward the diaphragm as the clamp is released and the purse-string suture is drawn taut around the advancing finger. In this manner very little blood loss will result. In our practice no more than three fingers are allowed to feel the valve (the first assistant before, the surgeon during and the first assistant after commissurotomy) and this only when conditions are ideal. If the patient's condition is less than



Fig. 7 The interior of the opened appendage repeatedly flushed with saline to remove all loose thrombotic debris.

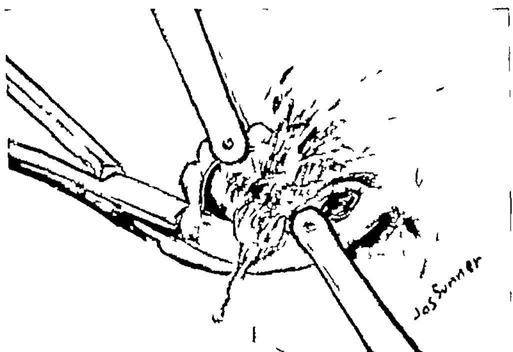


Fig 8 The atrial clamp is momentarily released to allow a gush of blood to escape from the left atrium outward through the appendage Thrombotic material that may have been caught by the clamp is removed in this manner Occasionally loose emboli floating freely within the left atrium have been expelled

ideal or if any thrombosis is found within the appendage, the surgeon'
only may be inserted.

No two appendages are exactly alike They may vary from a ve
nubbin to the size of a man's four fingers They may be narrow and
ated like the cecal appendix or large and S-shaped like the duodenal
even absent. When the appendage is small or absent, we routinely
the method shown in Fig. 9. The atrial wall surrounding the append
its site is widely purse-stringed with additional stay sutures placed
superior and inferior angles of the proposed incision The stay sutur
then be used as handles to apply traction against which the apper
clamp can grasp the atrial wall should this maneuver be necessary. In

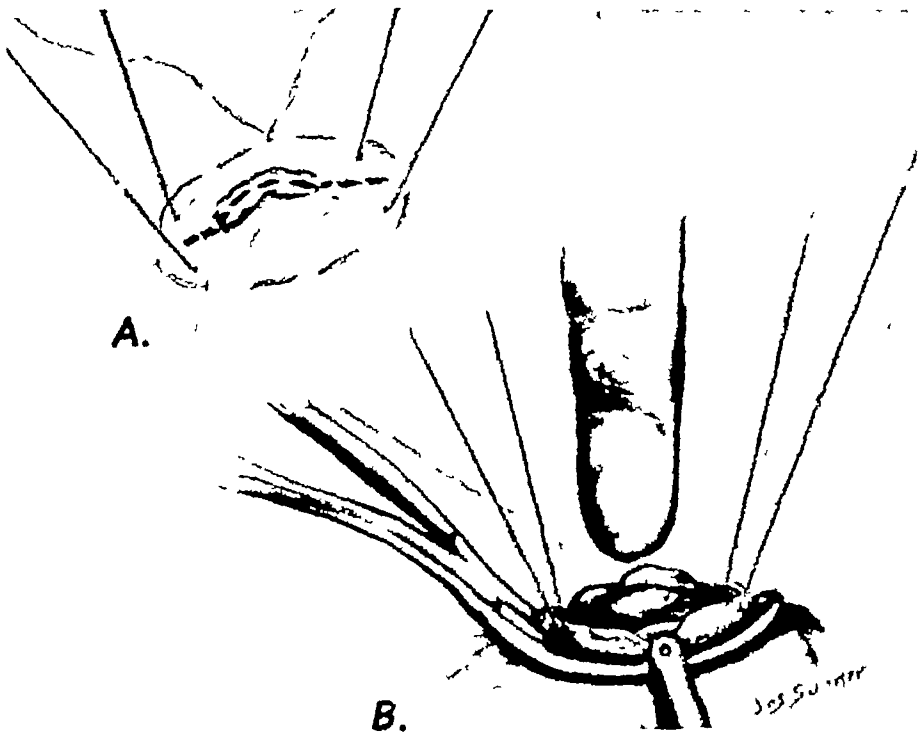


Fig 9 Method of entering the left atrium through a tiny appendage or th
of the atrium directly *A*, Purse-stringed atrial wall, stay sutures placed at the
of proposed incision. *B*, Stay sutures under traction for application of the atrial

tion, the stay sutures may be crossed should hemorrhage occur or if nec
tied in a crossed position in addition to tying the purse-string suture
incision is made in the usual manner directly through the tiny appendag
usually made slightly smaller than the size of the finger so that the tip
finger can be placed into the incision and gently and gradually screwe
the atrium without splitting the myocardium. Should there appear
danger of myocardial splitting at the angles the clamp is reapplied, a s
placed and tied across these angles to limit the size of the opening.
method has been found to be routinely satisfactory in all such inst
even when no appendage is present or when, by design, one plans to
the wall of the atrium directly. It is rarely necessary to employ any ty
plastic sleeve sewn to the atrium unless prolonged and intricate mane
are contemplated

Should the appendage contain thrombotic material or be completely obliterated by it, our practice even under these circumstances is to traverse its dissected lumen rather than make an additional incision in the atrium or use the pulmonary vein. Obviously all *loose* thrombotic material will be removed by direct dissection, multiple syringing and suction.

It is important not to attempt the complete removal of all thrombotic material within the appendage under certain conditions Two types of thrombotic material are readily distinguished and may be encountered in the same appendage or found separately The first and more dangerous is the loosely attached, granular, inspissated, clot-like material which is easily dislodged. All of this must be routinely removed. This type may occur alone or may

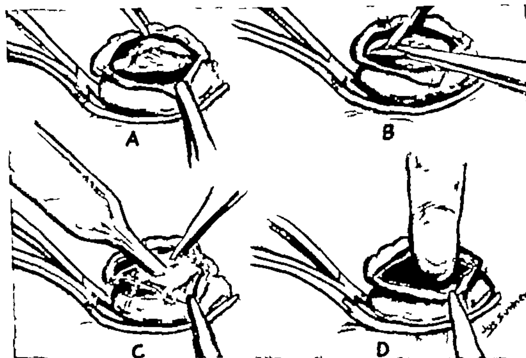


Fig. 10 Management of appendage obliterated by thrombosis *A*, Tip of auricular appendage incised revealing an inner thrombotic appendage Rather than to attempt dissection down either side of this thrombotic mass or its complete removal its tip is also incised (*B*), its interior cleaned of all loose debris (*C*), and the finger introduced into the left atrium through the doubly thickened appendage (*D*)

be enclosed in an envelope or inner appendage formed by a leathery, laminated layer of strong thrombotic material (Fig. 10*A*) It is preferable to incise this inner thrombotic appendage just as one originally incised the outer, natural appendage (Fig 10*B*) If one attempts to remove it in toto, there is danger of buttonholing and lacerating the appendageal myocardium in addition to creating a great deal more loose and friable fibrinous material than was already present. The interior of this inner thrombotic appendage usually communicates adequately with the lumen of the left atrium. With the protection of the proximal clamp considerable time can be spent removing all loose debris from the interior of this inner appendage employing direct dissection, extensive syringing, suction and release of the clamp to allow out-

ward bleeding (Fig. 10*C*). The inner and outer appendage walls are then grasped in the same forceps and the finger is introduced into the atrium just as one would traverse a normally clean appendage (Fig. 10*D*).

There are those who strongly advocate the routine use of temporary but intermittent bilateral carotid occlusion to protect the brain from emboli whenever thrombosis in the appendage or calcified valves are encountered. Such carotid occlusion may be obtained by external pressure on the carotid vessels in the neck or by surgical dissection and mobilization of the carotids within the mediastinum for the application of tapes or occluding instrument. The former method is ineffective and uncertain and may be dangerous, as the latter method most certainly is. Objections to this practice are as follows. Emboli to the brain may occur before or after such an occlusion is applied, for prolonged and constant occlusion cannot be employed throughout the danger period of appendage manipulation and valve reconstruction. Secondly, the carotid vessels themselves may be injured either during dissection or during the period of occlusion. Thirdly, thrombosis may and has been known to occur at the site of occlusion within the carotid vessels, completely defeating the purpose of the procedure. Fourthly, a measure of cerebral anoxia must occur during the period of occlusion, and this in itself may be dangerous and produce changes as bad or worse as those secondary to an embolic insult. Fifth, the time consumed to properly isolate and mobilize the carotid arteries may add unnecessarily to the overall time of mitral commissurotomy. These and other objections coupled with the fact that our cerebral embolic rate *without carotid occlusion* is lower than most of those who advocate carotid occlusion are ample reasons for our decision to refrain from the use of this procedure. We feel that if one accepts the responsibility for carrying out surgery in patients with a disease complex of this nature, the fact must likewise be accepted that a certain small percentage of cases will inevitably be complicated by operative emboli. More emphasis should be placed upon the proper handling and management of the thrombosed appendage and calcified valve Reference to our thrombo-embolic data as contained in Fig 11 will show that in 500 consecutive cases in which no form of carotid occlusion was employed isolated cerebral emboli occurred in 2 6 per cent

MANAGEMENT OF THE VALVE

With the finger in the left atrium the valvular conditions present can rapidly be assessed For most adequate exploration the finger should be introduced through the neck of the appendage beyond the second knuckle. In much less time than it takes to write, the following factors can be rapidly recognized The size of the mitral orifice, the position of this orifice in the valve cone, the location of the commissures, the consistency and pliability of the valve leaflets, the presence and position of calcium, the presence or absence of associated regurgitation, the presence or absence of associated pathology within the left atrium such as thrombotic material attached to the atrial wall proper or lesions of the atrial septum, all can be detected If there had been any question as to the presence of aortic valve disease, it might be well at this point to further explore this valve by palpating the aortic ring by pressure

on the aortic leaflet of the mitral valve. Perception of aortic valve pathology will be enhanced by counter pressure on the anterior surface of the aortic ring external to the heart, with the left hand squeezing and compressing the aortic ring between this hand and the intra atrial finger Additional degrees of valvular damage may be recognized by this maneuver

The average valve encountered presents an orifice the size of the tip of the finger or the circumference of a cigarette, occasionally not much larger than the head of a kitchen match stick. The cusp margins are thickened and rolled and an occasional bead of calcium may be felt at some point about the cusp margins The valve leaflets themselves are of the consistency of kid glove skin or thin shoe leather They will retain some degree of pliability and will balloon back onto the left atrium to greater or lesser degree with each ventricular systole. The mobility of the aortic leaflet of the mitral valve is usually

THROMBO-EMBOLIC DATA
(500 cases)

Pre operative	History of emboli		93 - 19%
	• Cerebral	61%	
	• Peripheral	28%	
	• Both	11%	
Operative (no carotid occlusion)	Thrombosis found at surgery of these only 52 cases (35%) with embolization		149 - 30%
	Emboli produced at surgery		22 - 4 4%
	• Cerebral	2 6%	
	• Peripheral	1 4%	
	• Both	0 4%	
Post operative	NO EMBOLI TO DATE IN ENTIRE SERIES oldest case 5½ years		

Fig 11 Thrombo-embolic data in first consecutive 500 commissurotomies (Glover and O'Neill) The text is self-explanatory

considerably greater than that of the smaller and more contracted posterior or mural leaflet. The entire atrioventricular ring or annulus may be contracted, sometimes up to 50 per cent, so that the total valve surface area is not much greater than a silver dollar If one imagines the valve cone to be the face of a clock and looks from atrium down toward the ventricle, the anterolateral commissure will be at eleven o clock and the posteromedial commissure at five o clock. Stated another way, with the patient in the true lateral position the anterolateral commissure is almost straight up toward the ceiling and the posteromedial commissure toward the floor The tip of the finger, with its palmar surface facing the region of the anterolateral commissure, is gradually introduced through the mitral orifice making constant pressure upward in the region of the anterolateral commissure. Simultaneously counter pressure

downward is made by the left hand on a sponge over the left ventricular myocardium (avoiding the circumflex coronary artery) (Fig. 12).

In favorable cases the commissure is felt to split and this dissolution of tissue (commissurotomy) is carried out to the annulus at the myocardium itself. One may have to knead the tissues a little to accomplish this complete separation. The finger is now passed down through this newly divided commissure in order to carry out any subvalvular dissection which may be necessary. The chordae tendineae are, as a rule, found to be greatly foreshortened and indurated and even agglutinated, so that instead of a considerable number of chordae tendineae being present one may only feel two or three on either side of the valve opening. The papillary muscles are hypertrophied and in



Fig 12 The right index finger applying pressure on the anterolateral commissure aided by counter pressure downward on the left ventricular myocardium.

close proximity to the cusp margins. By insinuating the finger downward the chordae tendineae in many instances can be freed from each other, sometimes lengthened by gentle massage, or they may be freed from any adherence to the myocardial wall (Fig. 13). The papillary muscles are most apt to be adherent to the myocardial wall and can be freed in a like manner. The sum total of this maneuver is to enhance the degree of valve motion and function

It is important to be sure that the commissure has been divided completely to the myocardial wall and that one can actually feel this wall flush with the outer extremity of the commissural separation Not infrequently the outer third or even half of the valve cone at its commissure lies almost parallel to the ventricular myocardium, so that the distal phalanx of the index finger must be crooked backward on the ventricular side of the valve to be sure that the commissure has been divided completely. The valve orifice should now accommodate two fingers side by side, and with good restoration of

valve motion in a favorable valve no significant regurgitation will have been produced.

It is desirable at this point to make pressure with the fingertip in the V of the posteromedial commissure, and not infrequently this commissure can be gradually divided for a distance of 1 cm. Occasionally by putting the fingertip on the ventricular side of this V and flicking it back into the atrium, it is possible to open a slightly more resistant posteromedial commissure. One should not persist in his attempt to open this commissure beyond 1 cm, for the danger of producing significant insufficiency in this area is very real. This is true because the chordae tendineae over the medial half of the mitral

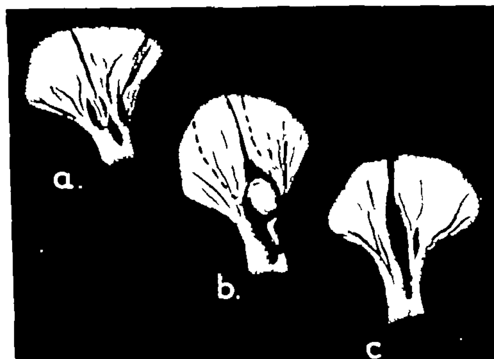


Fig. 13 Subvalvular dissection to achieve maximal mobility of cusps. *a* Illustrates the fused mass of thickened and shortened chordae tendineae which restrains motion of the leaflets even though the commissural fused cusps have been separated. *b*, Separation of this subvalvular mass can be accomplished by finger dissection in most cases. *c*, The desired result will be obtained in this manner (From Glover et al. Circulation, vol. 11, January 1955)

valve in disease tend to agglutinate and become adherent in one small area along the left ventricular outflow tract, so that in all probability one is not opening the true posteromedial commissure in the same sense as one opens the anterolateral commissure. Actually the surgeon is opening a commissural area just posterior to these clumped chordae tendineae, so that if this commissure is opened very far, a portion of the mural leaflet may be devoid of chordae tendineae and as such it will be a loose, flapping structure giving rise to considerable regurgitation.

An opening such as that described which will easily accommodate two loose fingers is most adequate and should lead to a highly satisfactory result. Unfortunately, however, in our experience an ideal commissurotomy such as this, accomplished without the use of a knife, is possible in only 25 per cent

of cases explored. Sometimes leathery, mobile tissues without calcification are harder to split in this manner than are more heavily calcific, fracturable tissues. In any event *pressure with the finger alone should be terminated when slight to moderate pressure has failed* lest a stronger avulsing pressure lead to a tear of a leaflet or a sudden rapid tear through the entire commissure out along the myocardium itself, tearing a portion of the leaflet from its attachment to the annulus. When such pressure is required, the proper use of a knife or guillotine, as we prefer, is an absolute essential.

Without disturbing the position of the finger in the auricular appendage the constricting purse-string can be loosened slightly and the curved guillotine knife, especially adapted to this posterolateral approach, is guided into

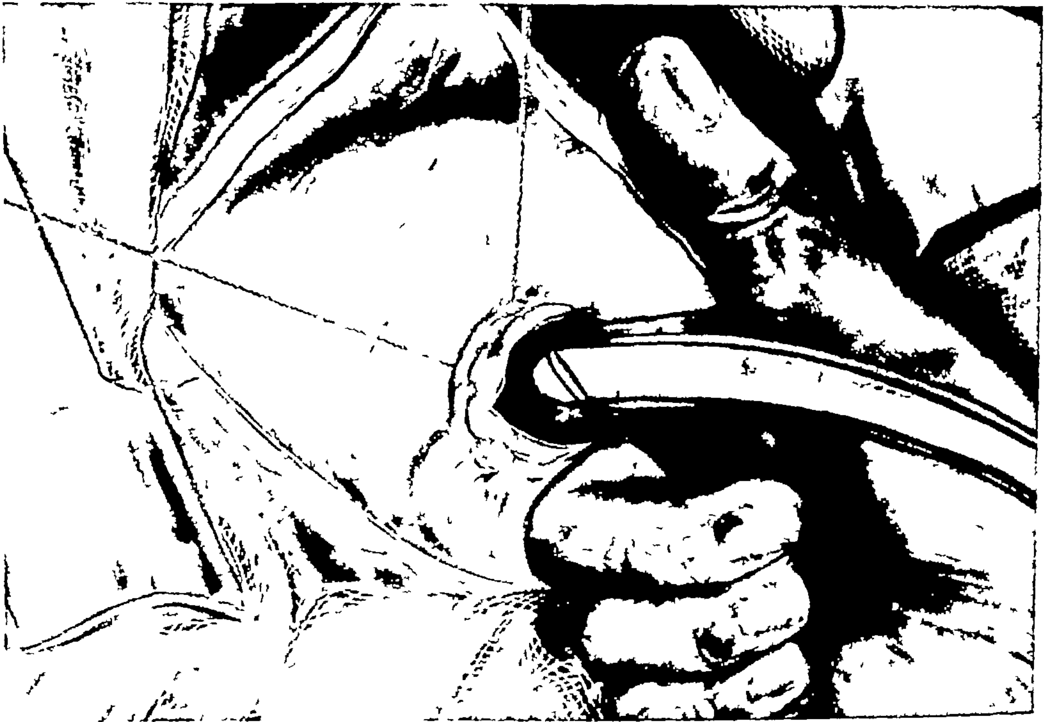


Fig. 14 The guillotine knife is slipped into the atrium along the palmar surface of the finger without removing the intra-atrial finger

the left atrium along the palmar surface of the finger (Fig. 14). The knife with the hook curved to the right and which when placed adjacent to the intra-atrial finger presents its concave surface toward the ceiling is the one used to open the anterolateral commissure (Fig 5*c*) The hook of this knife is completely dull and is not used for cutting but rather to engage the anterolateral commissure rendering it immobile at its orificial angle (Fig. 15). The knife, of course, is introduced into the atrium in the completely closed position so that no sharp surfaces are presented. The knife is kept in this closed position as it is introduced through the mitral orifice, being stabilized by the intra-atrial finger. At this point the guillotine blade is slowly withdrawn with the left hand so that it loses contact with the hook The indurated angle of the commissure will slip into the resulting gap (Fig 16) It is held in contact with the commissure by the intra-atrial finger. One complete cut is

made, incising no more than 0 5 to 1 0 cm. of tissue, i.e., the size of the cutting instrument.

By this maneuver, therefore, the tough, indurated rind of the cusp margin at the commissure, previously unyielding to finger pressure, is easily divided The closed guillotine is then withdrawn into the left atrium or removed entirely The remainder of the commissure, as a rule, is easily split out to the annulus now that the commissurotomy has been started in the right place (Fig 17) If additional unyielding areas impede the digital pressure, the knife



Fig 15 Finger and guillotine in position for opening the anterolateral commissure Insert shows commissurotomy completed.

is easily reinserted and the maneuver as described repeated. The dull hook of the knife can frequently be used with particular efficiency at the outer extremity of the commissurotomy near the myocardium. In this area a small half moon of tough tissue may prevent complete separation of the leaflets when finger pressure alone is used. Engagement of this tissue with the hook is a simple matter and the commissurotomy can easily be completed (Fig 18*c*)

Similarly the posteromedial commissure is opened using the left guillotine knife and directing the hook posteriorly It is again stressed that provided a complete anterolateral commissurotomy has been accomplished with a resultant orifice of two-finger size, the posteromedial commissure is opened but little. If, however, the anterolateral commissure cannot be opened satisfac-

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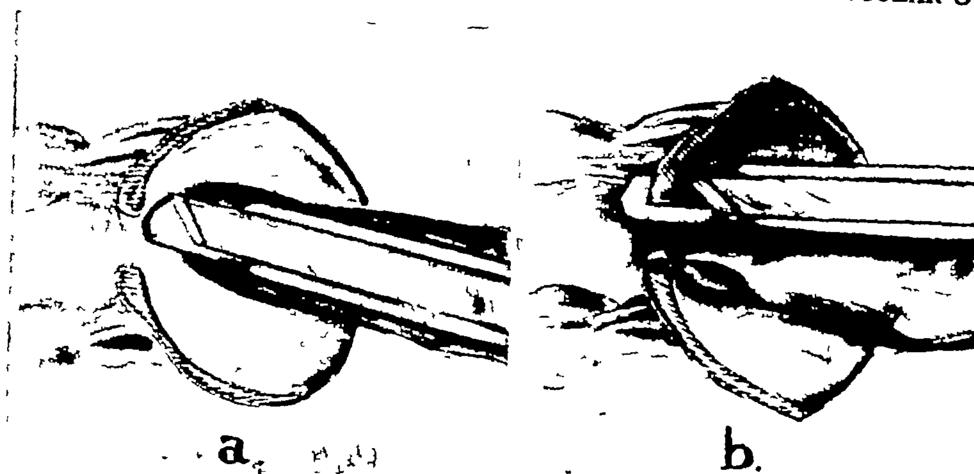


Fig 16. *a*, The guillotine is introduced into the mitral orifice in the close
tion *b*, The cutting blade is withdrawn, the hook falls in place engaging a
mobilizing the commissure preparatory to division of the tough and resistan
missural margin

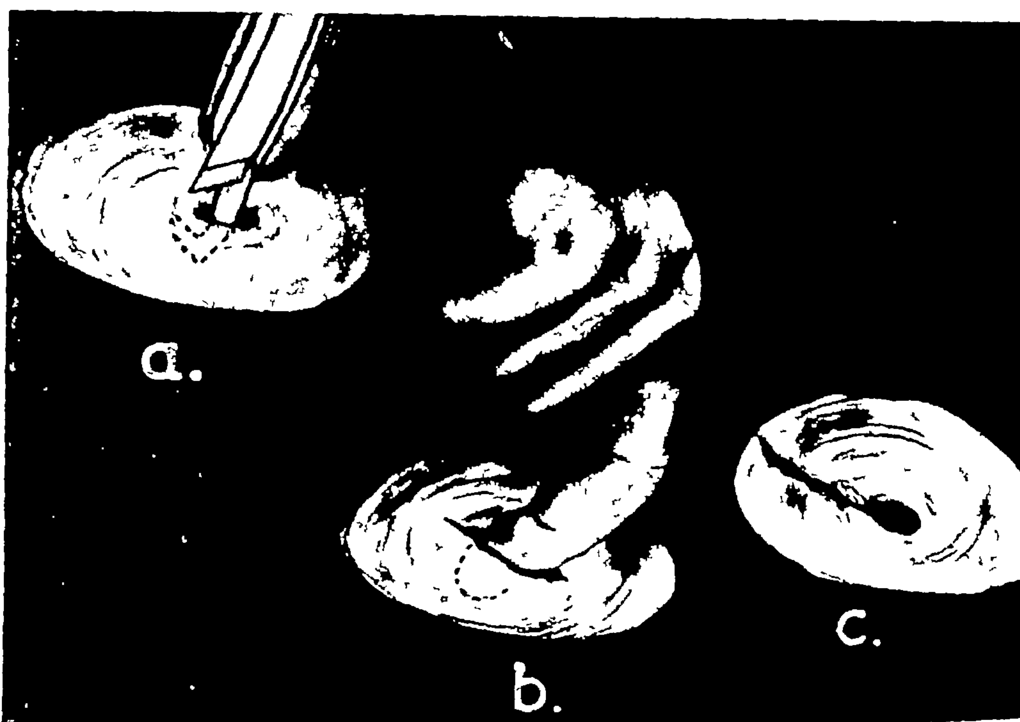


Fig 17. An adequate commissurotomy necessitates complete anatomic separ
of the fused valvular cusps from the stenotic orificial rim out to the annulus
This can be achieved best by initiating a cut through the thickened portion of
fused commissure at the rim of the orifice (*a*), and subsequently by either di
separation of the remainder of the commissure (*b*) or when necessary by mult
cuts with the hooked guillotine knife (From Glover et al Circulation, vol 11,
uary 1955)

torily, considerable effort must be applied to the proper and more compl
division of the posteromedial commissure.

To recapitulate, therefore, it can be readily seen that although an adequ
commissurotomy can at times be performed by the finger alone, it is pref
able to use a combination of the knife and finger. It is understood, th
that the finger and knife are not rival forms of surgical technique but rat

are complementary to each other in the proper performance of mitral commissurotomy We have used the combination of knife and finger in 75 per cent of our cases As a rough estimate it can be stated that in 25 per cent of the cases the finger alone, if used properly, may be adequate; in 50 per cent of the cases a more complete and accurate operation can be performed employing both finger and knife, in the remaining 25 per cent it is absolutely essential to use a knife for otherwise the operation cannot be accomplished It follows, therefore, that any surgeon proposing to perform a mitral commissurotomy must be equipped with a knife and must know how to use it if he is to properly

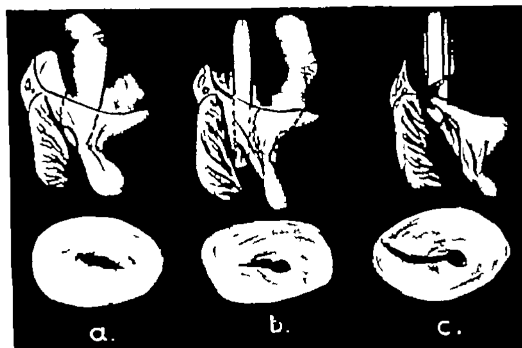


Fig 18 In some instances even though care is taken to attempt a separation of the cusps out to the annulus the false impression that this is achieved may be the result of "pressing" the relatively pliable edge of the membranous cone against the myocardial wall (*a* and *b*) This pitfall may occur in the course of finger dissection or even when cutting of the commissure is attempted with a straight bladed instrument. (*c*) The guillotine knife with the blunt hooked end is particularly valuable in assuring that the membrane be held and effectively cut. (From Glover et al. Circulation, vol. 11 January 1955)

discharge his responsibility to the patient. It need hardly be repeated that, following the division of the commissure by whatever technique, subvalvular finger dissection of chordae tendineae and papillary muscles for more complete mobilization and function of the valve leaflets is essential.

CLOSURE

When the surgeon is satisfied that the valve has been opened properly, the finger is withdrawn from the left atrium as the clamp is reapplied. The previously placed purse-string suture is now tied. The appendage distal to the purse-string suture is amputated and sent to the laboratory for histologic study The stump of the appendage is oversewn to insure hemostasis (Fig 19) The heart and pericardial sac are generously flushed with saline to remove

all blood or clotted debris. The diastolic thrill near the apex of the left ventricle will have disappeared. The ascending aorta and aortic ring are again checked to be sure that improved left ventricular function with its resultant increased output has not now disclosed evidence of aortic valvular disease previously unrecognized. The pericardium is partially closed leaving a generous portion open, however, in the region of the stump of the appendage to insure pericardial drainage into the pleural space and to prevent a compression pericardial effusion (Fig. 20). It may be well to make a small opening into the pericardium just above the diaphragm so that more dependent drainage of the pericardial sac can be maintained. The pleural space is likewise flushed and cleaned, the lung expanded and a lung biopsy obtained The pleura is drained by catheter and the chest wall closed in the usual manner.



Fig 19 The appendage is amputated distal to the ligating purse string The stump is securely oversewn to insure hemostasis

The average case will have tolerated the procedure well, blood loss from the cardiac chambers will not have exceeded more than several ounces, and the operating time from skin to skin will rarely exceed one hour and thirty minutes. The patient at this point is awake, although a little groggy. He is asked to talk, to wrinkle his eyebrows, to whistle and to move all four extremities. If these maneuvers can be accomplished, no cerebral embolus has occurred. Should there be obvious evidence of a hemiplegia secondary to a cerebral embolus, the patient is immediately subjected to a stellate block on the side of the brain lesion. The stellate ganglion is kept blocked for the next 48 hours Should there be obvious evidence of a saddle embolus at the bifurcation of the abdominal aorta or a major peripheral embolus, embolectomy is carried out at once. After suitable tracheobronchial toilet the patient is returned to his room.

The average patient is ambulated from the third to the sixth day and discharged from the hospital in twelve to fourteen days He is cautioned to remain completely inactive for the ensuing six weeks and only then to gradually increase his activity It is likewise explained that the full and final result of his surgery may not be complete for from six months to a year and that he may well make greater subjective progress in the second six postoperative months than in the first six months He is continued on a maintenance dose of digitalis and a low salt diet for at least six months, at which time these measures may be readjusted at the discretion of his attending physician Rarely are mercurials needed during the postoperative period despite their use preoperatively

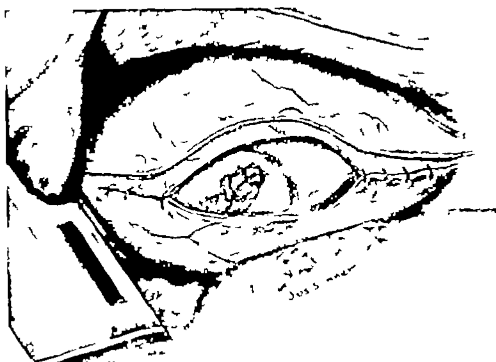


Fig 20 Partial closure of the pericardium to allow for drainage of pericardial fluid into pleural space This avoids danger of compression pericardial effusion

MITRAL INSUFFICIENCY

The major and most distressing complication in our series of commissurotomies leading to an early demise or a prolonged downhill course has been the inadvertent production of significant mitral insufficiency or the aggravation of previously existing associated insufficiency Although our cardiologists have valiantly attempted to refer to surgery only those cases of pure or greatly predominant mitral stenosis, not infrequently the exploring finger will encounter considerably more regurgitation than was suspected clinically In other cases even when the commissurotomy has been properly performed, significant degrees of insufficiency have developed owing to the peculiar nature of the valvular pathology Since the initiation of this type of surgery, we have been aware of the fact that complete rehabilitation of many of these patients will not be accomplished without some method for the control of the regurgitation present. At no time, until recent developments mentioned

below, have we knowingly accepted cases for mitral surgery when the regurgitant factor was obviously predominant, for no *effective* techniques for its correction have been available.

In 1950 and 1951 we observed that the degree of insufficiency appreciated by the finger in the left atrium could be greatly diminished or even abolished by manual constriction of the mitral annulus between the left index finger and thumb. This prompted us to attempt the development of measures designed to constrict the atrioventricular ring. During the next two years on eight occasions, we attempted to apply this principle of annular constriction by imbricating the pericardium in such a way as to apply as much pressure as

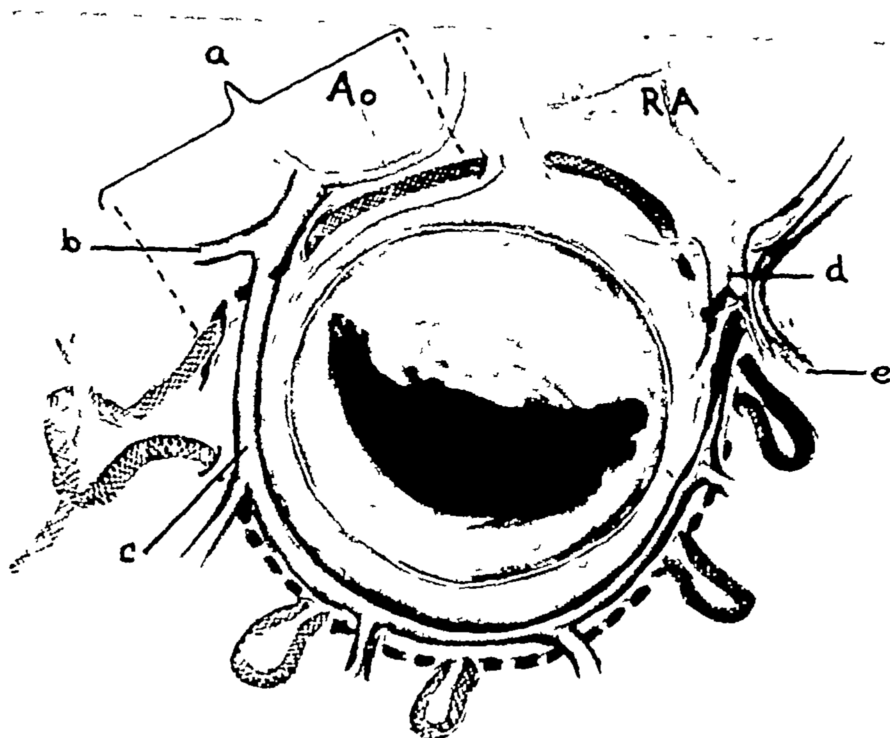


FIG. 21. Diagram of the mitral valve ring showing the relations of the circumferential suture *a*, Segment of suture lying in the transverse sinus and beneath the circumflex coronary artery This segment of suture is covered with pericardium to prevent the suture from eroding through the base of the atrial appendage *b*, Anterior descending coronary. *c*, Circumflex coronary *d*, Coronary sinus *e*, Posterior descending coronary.

possible to the external aspects of the mitral ring. The impression was gained that some benefit resulted from this maneuver. On three other occasions when associated aortic insufficiency was noted the aortic ring was carefully injected with a sclerosing solution of sodium diacetyl phosphate and again the clinical impression was that of possible improvement This led us to a study of the anatomy of the mitral ring in a series of cadavers obtained from the postmortem room at the Episcopal Hospital, Philadelphia. The report of these studies and the proposed method of partial annular constriction of the mitral ring, so-called "mitral purse-string," have been previously published. For the past two years the facilities of our Cardiovascular Research Laboratory at the Presbyterian Hospital in Philadelphia have been devoted to the study

of valvular insufficiencies using the principle of annular constriction as the basis of our therapeutic endeavors The concept of possible universal application of purse-stringing valvular rings was thereby originated, has since been developed experimentally and more recently applied clinically Our associate, Dr J C. Davila, will present in this symposium some of the clinical material and results that have been obtained to date. It would appear that provided the mitral leaflets retain some degree of pliability and motion, constriction either of a dilated mitral ring or even a ring of relatively normal size will result in coaptation and competency of these leaflets without the production of a stenotic effect (Fig 21) Should this procedure withstand the test of time, an adjunct of great magnitude will be added to the operation of mitral commissurotomy, to say nothing of its great application in those myriads of patients who heretofore have not even been considered as candidates for mitral valve surgery because of the high degree of mitral insufficiency present

CONCLUSION

The aims of mitral commissurotomy and its techniques have been described in detail because it is felt tha much misconception and misinformation regarding the performance of this operation has been rampant among the profession to date. Anything less than the above described commissurotomy is not considered to be ideal, although, to be sure, the distortion of the valve tissues, the imbedded calcium, the shortened and fused chordae and papillary muscles may prevent the operator from accomplishing this desired goal in a number of cases It must, however, be the pathologic changes of the valve tissues themselves that dictate the limitations of the operative procedure and not the surgeon's ignorance of what constitutes an ideal commissurotomy Within the past three years, the performance of intracardiac valvular surgery of this type has become the ambition of almost every surgeon who has ever had occasion to open the thorax, and, as a result, commissurotomies have been attempted by many operators who have had little or no experience in this field, and incomplete understanding of the fundamental surgical goal. For example, there are many who merely thrust the index finger through the tiny stenotic valve orifice, feel the tissues split to a degree on either side and then terminate the procedure, feeling that a proper commissurotomy has been performed. Under these circumstances, the valve leaflets have not been adequately separated and a considerable bridge of fused tissues remains to prevent adequate restoration of valve motion (Fig 22) Most certainly such an incompletely divided commissure may act as the locus for reagglutination, either because of the proximity of the raw cusp margins, or from the deposition of fibrin and particulate matter leading to thrombosis Under these circumstances the patient's functional improvement will be short lived, and such results may dampen the enthusiasm of both physician and laity for an operation the value of which, when properly performed, is unquestionably of considerable magnitude. On the other hand, when the commissures are widely opened, this state of affairs will result but rarely An analysis of 35 autopsied cases in our series, death having occurred from one day to three and one-half years postoperatively, has failed to reveal a single instance of valvular restenosis

Therefore, an understanding of these fundamental principles in the technique of mitral commissurotomy is obviously essential not only for the proper performance of the operation but also for the intelligent future evaluation of the valvular status of patients so treated.

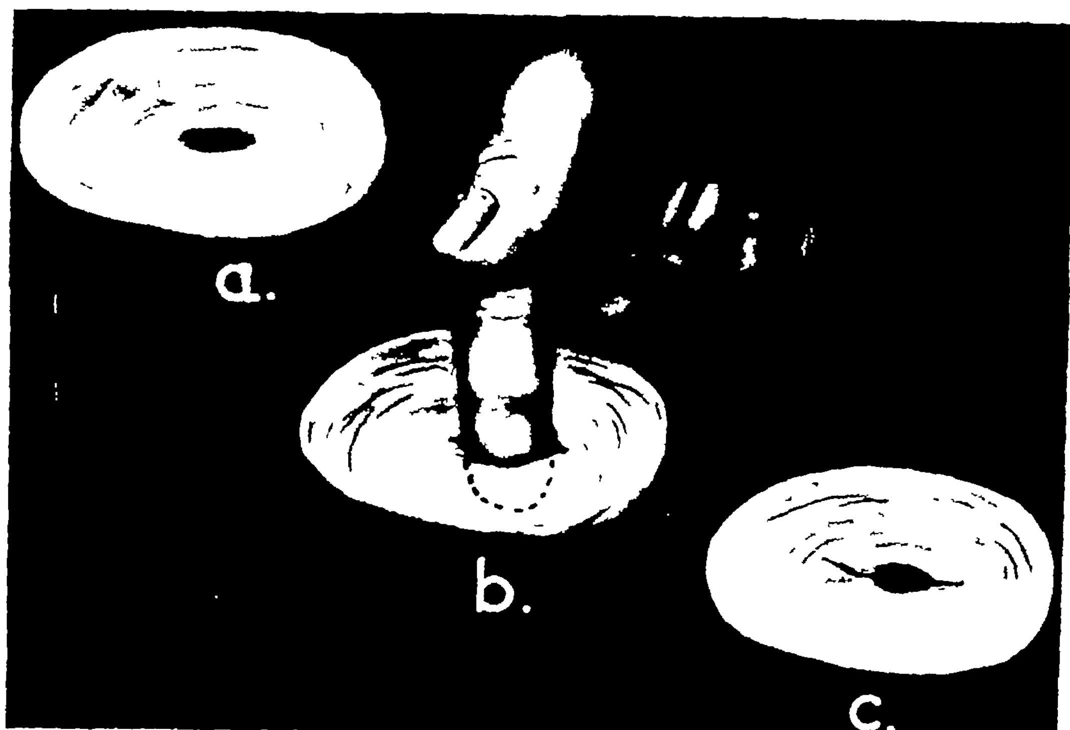


Fig 22 An inadequate commissurotomy A simple thrust of the finger through the stenosed orifice (*a*) does not constitute acceptable valvular surgery. As the finger is thrust through the valve (*b*) a sensation of "give" may be felt This is due to slight tearing at the angles, but if the procedure is limited to this maneuver the result will be an inadequate increase in the size of the orifice and little, if any, improvement in valvular motion (*c*) (From Glover et al Circulation, vol 11, January 1955.)

DISCUSSION

Clarence Crafoord (*Stockholm*)

Dr. Glover has covered the technical aspects of mitral stenosis in such an excellent way that there is not very much to add. However, there is one cause of mitral stenosis which has not been touched upon at all during this symposium, and I should like to discuss it

Figure 1, a selective angiocardiogram, shows within the area of the left atrium a big part of the atrium that is not filled with contrast medium This case was sent to us as one of mitral stenosis with episodes of fainting. The clinical picture was not quite typical, so we proceeded with various investigations, including direct atrial puncture with pressure measurements, and after angiocardiography we finally got the answer to what was wrong in this case This is a case of myxoma of the left atrium which has caused mitral stenosis.

In cases of mitral stenosis, specifically those with calcification and the very difficult ones, I believe that open cardiac surgery will have its place in the future, and can be performed then with less risk than it can now.

In this case nothing else than open cardiac surgery could be of any help This patient was a woman between 40 and 50 years of age. She was on extra-corporeal complete circulation and oxygenation for thirty minutes during the time of intervention, when we opened up the left atrium very widely and during which time we compressed the mitral orifice.

I had my assistant compress the left ventricle so that it completely closed from the time the artificial circulation was started. Then I opened up the atrium, and it was possible to remove this huge tumor practically in one piece. Some of it could not be taken out of the incision, although the incision was very large. It had to be broken up in three different parts After that was done the atrium was flushed and closed, and the patient had only minor complications in the postoperative period A little less than one year after the operation she is in very good condition.

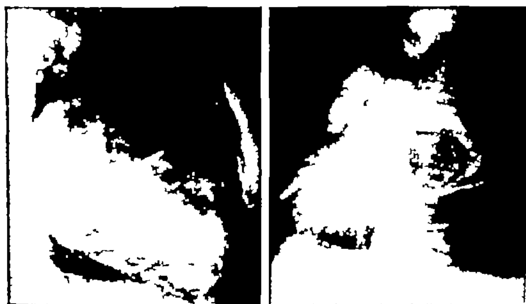


Fig 1 Angiocardiogram showing large filling defect in the left atrium which proved to be an intracardiac tumor

Fernando Tricerri (*Buenos Aires*)

I feel, as Dr Crafoord felt, that very little has to be added to Dr Glover's excellent paper I would like to make a comment on three points

First, the surgery that is being done on mitral stenosis has several names—valvuloplasty, commissurotomy and valvotomy Whatever name you use, I think the surgeon should keep in mind clearly that if he has to do a complete job this includes section of the commissures, treatment of the papillary muscles and also the chordae tendineae.

The second point refers to the emboli. In 200 cases we have had 5 cases of cerebral emboli. Four of those cases were fatal and were all patients with atrial fibrillation, calcified valves, and old age. One patient who recovered completely had sinus rhythm and a calcified valve.

The third thing I want to say concerns the use of hypothermia in very severely ill patients with mitral stenosis We have used hypothermia in 10 cases in this group, following the suggestion of Bigelow, using the cooling

technique of Swan. Temperatures were reduced to 30 degrees. All the patients were in cardiac failure and did not respond to medical treatment at all. They all did well.

David P. Boyd (*Boston*)

Since I am associated with an extremely conservative group of cardiologists, our series has been small. Although I am sure that many hundreds and perhaps thousands of cases of mitral stenosis have been seen at the Lahey Clinic, our series is just under 100 cases.

There is one point about early postoperative technique that may well be of sufficient value to report. I think Dr. Glover has been extremely fortunate in his low incidence of late embolism. We have had 8 cases of embolism in 93 mitral valve operations, and of these 2 ended fatally, one being a residual hemiplegia and the other a late recovery.

I want to speak briefly about the last two patients. Neither of these patients had calcium on the valve or clots in the atrium, and we assume that the appendage was entirely obliterated by the procedure. The first patient was operated on in the afternoon. The following morning, she was hemiplegic. In preparing to do a stellate ganglion block, I observed that the left common carotid was not pulsating. She was taken immediately to the operating room, and clots were extracted from the common carotid artery, resulting in good blood flow. Eighteen hours went by, and although the patient did respond after this procedure, and seemed to have some movement of her leg, she expired that afternoon.

After this experience we determined that we would watch the carotids carefully in these patients. It just happened that we had a similar experience a week or two later, but it was picked up a few hours postoperatively. The patient was taken back to the operating room immediately. Again the left common carotid artery was not pulsating. A clot was removed from the artery at the junction of the aorta, and the patient made a complete recovery.

All of these patients had carotid occlusion. In discussing this matter of carotid occlusion with our neurosurgeons, I found that they regard it with considerable alarm from the standpoint of spasm of the carotid system. When I remind you that neither of these later two patients had clots or calcium in the atrium, and yet developed thrombosis in the left common carotid artery, we have to assume that perhaps the manipulations associated with this maneuver of carotid compression may have something to do with it.

I leave this thought with you simply to have you think that the carotid should be watched continuously through the surgery and until the patient responds afterwards.

Ormand C. Julian (*Chicago*)

I would like to mention one point in technique which we have thought important.

One year ago, upon entering an atrium, we were surprised to find a large, soft, intact clot lying above a very tightly stenotic mitral valve. We introduced the glass type suction along the side of the finger under very strong suction,

aspirated apparently all of this soft clot so that postoperatively, having had the mitral valve opened, the patient did not have an embolus

Since that time, during the past year when we feel that there is some chance of embolus, we have this strong suction apparatus ready We have made it a habit to clamp the suction tube so that the immediate aspiration will be extremely strong Once since then we have encountered a favorable case for its use. Other cases with firm clots within the atrium, well adherent of course, have not been disturbed in this fashion because we would despair of getting all of the clot.

I think in the presence of a soft clot this procedure merits consideration.

Marceau Servelle (Paris)

Since 1950, we have performed 220 commissurotomies for mitral stenosis, and the mortality rate has been 5 per cent. Almost all of our patients have been catheterized before and after operation in the services of Professor Soulie at the Lariboisière Hospital

In the early days, we performed most of the commissurotomies with the finger and some with the guillotine of Bailey, Glover and O'Neill. We have also used Brock s knife and the Dubost dilator Our impression then was that in about 50 per cent of cases, digital fracture was sufficient. In using the various knives invented for commissurotomy, we always fear the production of regurgitation. Dubost's dilator makes a good opening, but it is apt to catch the valves, and each time we use it, we enclose it in a rubber finger cap which is a hindrance.

It appears to us that if relapse is to be avoided and long-term results insured, it is necessary to open the commissures both medially and laterally, up to the insertion of the valves, without the production of regurgitation. We have designed a mitral dilator which will accomplish this, without danger of catching tissue when it is inserted or withdrawn Since using the instrument, we have had no accidents and no mitral regurgitation.

Charles Dubost *(Paris)*

We think that the finger is the best instrument for dividing the mitral valves which have become fused together, but its possibilities are found to be restricted. In the majority of the cases in our series, the finger was not able of itself alone to split the commissures, and so at the beginning of our practice we have tried out different forms of valvulotomes and different models of guillotines which were recommended to us

We have rarely been able to obtain, with safety, the results hoped for, in an easy way Either the atrium was of too modest caliber to permit the entry of the index finger provided with an instrument, or it was difficult to find out exactly the position of the commissures so as to make a start on their separation. And so we were quickly directed towards the search for a different method, with the intention of obtaining a dilatation of the valves by an instrument which would replace the finger We had to have the proof that the required instrument would be able to split the commissures without involving damage at the level of the valves, without tearing off one or other

of their suspending elements. We verified the merits of our hypothesis on a certain number of anatomic specimens. The opening had been made, in two cases, in the axis of the commissures, in the middle of the fan-shaped arrangement of chordae tendineae.

From this point, we decided to make a special dilator the size of which would be very much less than that of the index, and which would have at its extremity two branches which would be capable of opening on the exertion of pressure to the two handles of the instrument.

A second model was also constructed which was shorter and of slightly larger caliber which would allow, in the eventuality of an atrium of posterior

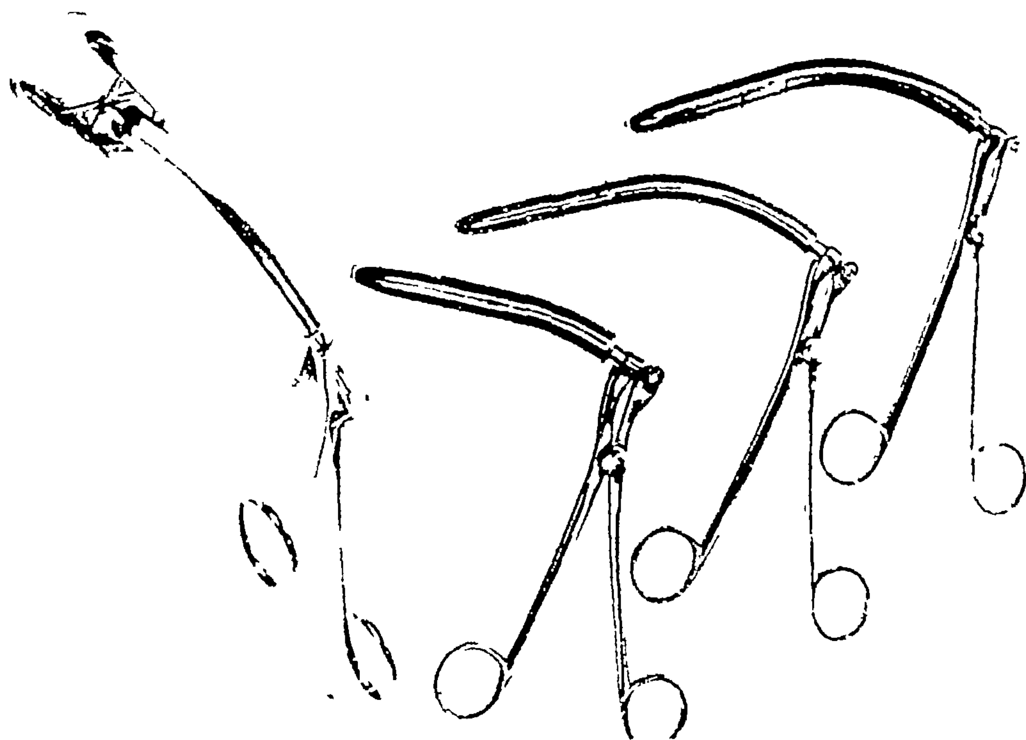


Fig 1 The different types of the mitral dilator

origin, overcoming the difficulty and obtaining a commissural separation. Finally a third model was made which was longer and of lesser caliber. This instrument can be indispensable in certain cases, especially with children.

METHOD OF INSTRUMENTAL DILATION

We routinely use the axillary thoracotomy with resection of the fourth rib The opening should be extended well into the armpit so as to allow a wide thoracic gaping and consequently the easy maneuver of the heel of the instrument.

The atrium is controlled by a single purse-string suture in conjunction with a Rumel-Belmont tourniquet

Diagrammatically three eventualities can occur.

1 Either the auricular appendage is of a good size with a wide base of attachment to the atrium, in which case the introduction of the index presents no difficulty.

2. Or the atrium is small and the use of the index would seem difficult, and so it should be replaced by the little finger

3 Or the atrium is absolutely unusable which makes it necessary to penetrate through the wall of the atrium itself

After having tried digital commissurotomy, the index is withdrawn from the atrium and is replaced by the metallic dilator This instrument is inserted in the same way as the index, by loosening progressively the clamp of the atrium and at the same time tightening the purse string suture by means of the Rumel-Belmont tourniquet The smooth cylindrical form of this instrument, devoid of all asperities, enables its insertion to be effected without the loss of a drop of blood

From this moment, as the position of the mitral stenosis is known, the extremity of the instrument is directed softly towards it, without any forcing as the dilator should penetrate into the preliminary hole created by the index,

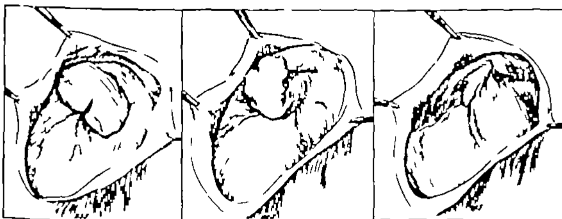


Fig 2 *A B, C,* Auricular appendages of different sizes

of its own accord Necessity to overcome a resistance by force would signify that the extremity of the dilator was not in front of the orifice to be entered, but was in contact either with a valve or with the mitral ring

With a little practice, and knowing that the mitral orifice is very highly placed when in an operational position, the direction of the instrument can be carried out without difficulty as far as this orifice is concerned. In fact it penetrates more quickly and more deeply than is wished for, as if sucked in by the ventricular diastole. It should therefore be withdrawn for two or three centimeters in such a way as not to run the risk of opening the expandable branches in the deep surface of the valves, for if this occurred it would, without any doubt, entail the destruction of the suspensory apparatus and perhaps prevent the extraction of the instrument. (We have recently built a new model which prevents this accident.)

As soon as the impression is gained that only the extremity of the dilator is in the right place, the application of pressure, most often slight, on the two branches suffices to split the commissures The fact that it is not necessary to make the greatest opening must be insisted upon, as a half of the possible maximum is, more often than not, quite sufficient to obtain the

desired commissurotomy. Therefore in order to avoid opening the dilator to the full, it is as well to place a finger within the separation of its branches in such a way as to limit their opening.

The instrument is then closed and withdrawn from the ventricular cavity and at the same time the clamp is replaced at the cavity base.

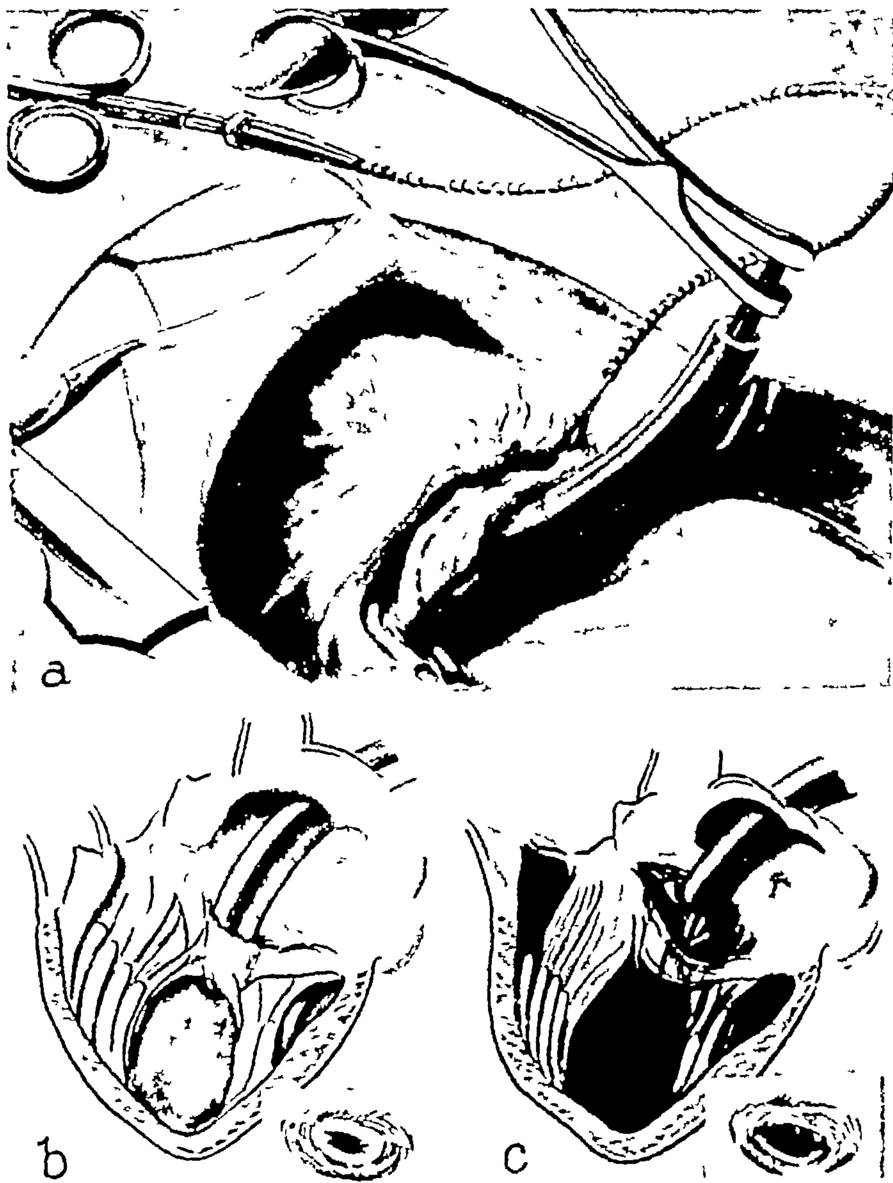


Fig 3 The mitral dilator (*a*, *b*) replaces the index finger, any loss of blood being avoided by the use of the tourniquet The dilator is opened (*C*) by pressure on its two branches

These manipulations take longer to describe than to carry out, as only a few seconds are required to insert the instrument into the mitral orifice and open the branches This speed is, in fact, necessary if the obturation of the atrioventricular orifice for more than two or three systoles is to be avoided

The index is then reinserted in order to verify the quality of the result obtained Sometimes the two commissures have split right up to the mitral ring, which means a perfect result as the opening corresponds to the width of about three fingers. At other times the anterior commissure is divided to

the mitral ring but the posterior commissure does not split completely, and a residue of only a few millimeters remains In any case we now insist on commissurotomies exceeding two fingers in width.

RESULTS

During the course of the last fifteen months, 230 mitral stenoses have been operated upon in the Broussais Hospital, in the department of cardiac surgery directed by Professor F d'Allaines Out of this number we have had 75 digital commissurotomies, and 155 commissurotomies carried out with the aid of the dilator

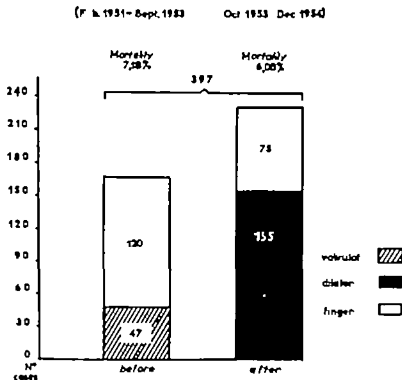


Fig. 4 Comparative study of mitral stenosis surgery before and after adoption of mitral dilator

These 230 interventions have given us 11 deaths, of which only 6 can be directly imputed to the dilator, either because this instrument may have been responsible for a perforation of the atrioventricular ring, or because, inserted too deeply, it was not able to be extracted from the ventricular cavity The other 5 deaths were due to postoperative complications (pulmonary embolus, hemopericardium, heart failure, cerebral embolism)

We were able to verify the efficacity of the dilator instrument on the anatomical specimens, and we noted the exact passage of the separation of the valves on the level of the commissures, causing thereby no damage whatever at the level of the subvalvular suspensory elements The anatomic results have been excellent in 75 per cent of the operations carried out with the aid of the dilator The opening obtained has always been more than two fingers, sometimes reaching up to two and one-half and three fingers, without there appearing the slightest systolic regurgitation in any one of them, regurgitation

which might have been due either to a valvular wound, or to the tearing of the suspensory elements of the valves. Moreover, we have confirmation that the disappearance of the signs of auscultation of mitral stenosis is maintained completely, although of course the first operations by dilation do not go further back than fifteen months.

155	DILATOR RESPONSIBLE FOR 6 DEATHS		MORTALITY IN DILATED PATIENTS	
	Perforation of the ventricular wall	3	Hemopericardium	1
	Rupture of a papillary muscle	1	Saddle embolism of aorta	1
	Rupture of the left atrium	2	Cardiac failure	2
	6		Pulmonary embol	1
			5	
TOTAL MORTALITY 11 (7%)				

Fig. 5 Mortality in 155 cases since the use of the mitral dilator (fifteen months)

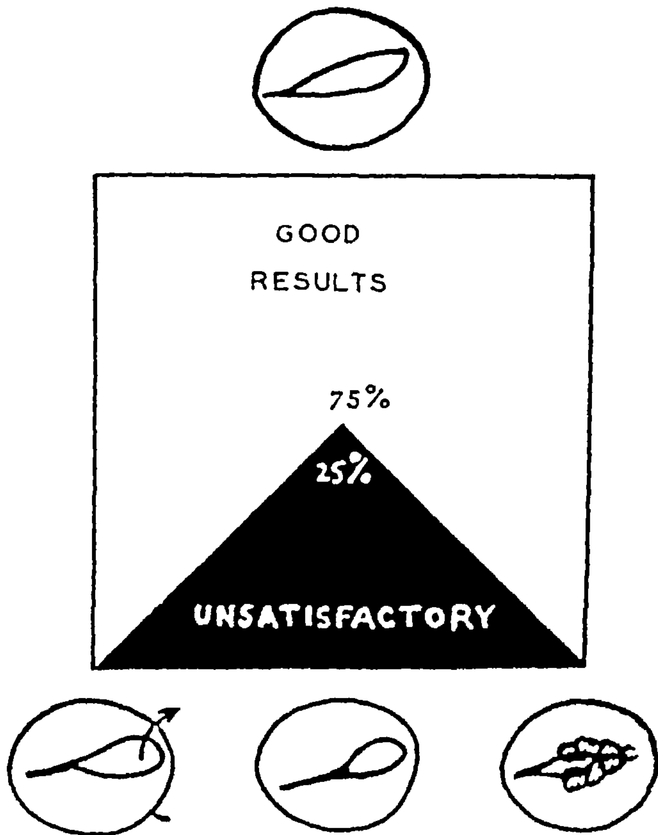


Fig 6 Results of mitral commissurotomy with mitral dilator

Finally, we have used this dilator instrument for an operation of a case of tricuspid stenosis in association with a mitral stenosis. The mitral commissurotomy was carried out by instrumental dilation through a left thoracotomy, in a first step, and in a second phase, following one month later, the tricuspid commissurotomy was then achieved (Fig 7).

The intervention revealed to us a form of stenosis which was very narrow and which split only incompletely with the finger The use of the dilator allowed us to carry out a commissurotomy of two and one-half fingers at the expense of only two commissures of the tricuspid, without any regurgitation

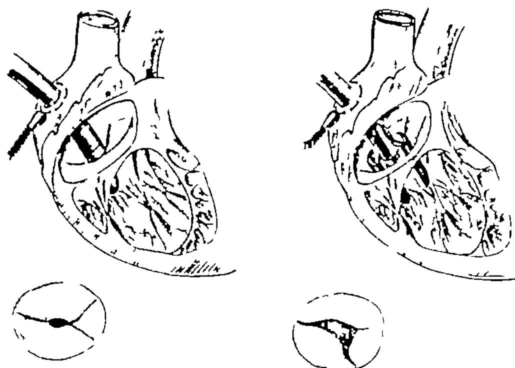


Fig 7 Tricuspid stenosis successfully dilated by use of the mitral dilator

We believe that a dilator instrument with three branches would have obtained a total result in this case, as it would probably have split the third commissure The effects of this operation were very favorable and the patient is today considered cured.

THE SURGICAL TREATMENT OF MITRAL INSUFFICIENCY

DWIGHT E. HARKEN (*Boston*)

INTRODUCTION

Our treatment of mitral insufficiency remains unsatisfactory. It is the uncomfortable discussion of what we've learned that constitutes my material here. Most of this has already been published, but there may be time for a few additional comments on further considerations of the pathology of the condition and possible future operations.

The surgical management of valvular diseases has fostered clear definition of life cycles. If we can recognize the terminal phase of a disease and its pure form, appraisal of therapy may be easier. In mitral stenosis it was possible to classify and define, among others, the "terminal phase." That it was in fact the end phase was attested by the nineteen "Group IV" patients in the first year of our work, who refused surgery. Seventeen were dead within the year; 15 within six months

In aortic stenosis we can also define the terminal phase. Ellis did a retrograde study of 100 patients with aortic stenosis who had come to autopsy He showed that left ventricular failure and atrial fibrillation were grave signs and, when these were followed by cardiac pain or syncope, death followed within weeks or months This is consistent with your clinical experience. During the past four years, 34 patients to whom we have recommended surgery did not accept for various reasons *Thirty of these 34 were dead within six months*. Indeed, in aortic stenosis, "it is later than you think."

Clinically and hemodynamically there is some similarity between terminal aortic disease and terminal pure mitral insufficiency. Left ventricular failure is common to both. Many patients with so-called "mitral insufficiency" have from the outset primarily myocardial disease and, in the symptomatic phase, all pure mitral insufficiency has some factor of left ventricular failure. This is quite unlike mitral stenosis.

In short, a terminal phase of mitral insufficiency probably can be defined. It is manifested by persistent and progressive pulmonary and peripheral congestive failure in spite of the full cardiac regimen. At this point, few patients spontaneously reverse and recuperate.

Far be it from me to suggest that surgery forever be restricted to such a poor group Indeed, many of them are beyond the point where the valvular

A portion of this work was supported by a grant from the National Heart Institute, Public Health Service

disease is very significant. However, if these people can be improved, the experimental corrective method is good. If the patients are not improved, however, it may not mean that the method is bad A good technique might even be overlooked, but that is the chance to be taken. In such a group, risks incident to unexpected complications of the experimental corrective technique are justified and can be accepted. I shall show you that even under these conditions there can be definite, though poor, salvage.

There is another aspect of evaluating techniques for correcting mitral insufficiency We must confine our study to *pure* mitral insufficiency Such a condition does exist. Virtually any attendant degree of stenosis invalidates appraisal of improvement. This is apparent from our three-year follow-up of our first 500 valvuloplasties Of patients with mitral stenosis and mild insufficiency, 82 per cent were improved. Of patients with mitral stenosis and moderate to marked insufficiency, 63 per cent were improved. Thus, the necessity for confining this study to terminal patients with pure insufficiency becomes apparent.

MORPHOLOGIC PATHOLOGY

In addition to the pathologic patterns that we have presented before, I should like to add another dimension. This dimension is a longitudinal one involving herniation of the valve complex and annulus toward the atrium. However, let us review the standard patterns first.

In mitral valvular disease a whole spectrum of pathologic states, ranging from pure stenosis to pure insufficiency, exist. Very little is known about the pathogenesis of these lesions, but it would appear reasonable to assume that stenosis occurs when, in the bullous phase of acute rheumatic valvulitis, the edges of the leaflets are forced together and adhere. This fusion probably progresses from the periphery where the range of motion is least and gravitates centrally until significant obstruction occurs It is also conceivable that broad zones of swollen leaflets pressed into apposition can seal together as a relatively acute process Such a sequence of events would result in mitral stenosis.

It is not difficult to conceive of a less intense inflammatory reaction or even a different type of reaction resulting in scarring, shortening and contracture of leaflets and chordae tendineae without causing them to adhere. This would produce an incompetent valve. In our experience the end result of such a pathologic process can be classified in five general patterns

TYPE I ABSOLUTE LOSS OF SUBSTANCE This is the basic form mentioned previously wherein the rheumatic process has attacked the leaflets directly and the resulting contracture prevents them from closing in systole. Because of its important function in the left ventricular outflow tract, damage of the aortic leaflet results in very serious incompetence (Fig 1).

TYPE II RELATIVE LOSS OF SUBSTANCE. This occurs either as the result of rheumatic carditis which attacks the annulus directly or because of generalized left ventricular dilatation secondary to myocardial disease and failure. As the annulus enlarges, the valve leaflets, otherwise adequate in length, fail to approximate, and regurgitation results This mechanism includes those

forms of incompetence often referred to as "functional insufficiency" (Fig. 1).

In our experience almost all *pure* mitral insufficiency results from a combination of the aforementioned two mechanisms. Conspicuous features of pure insufficiency are the absence of calcification and the resultant free mobility of the leaflets. With mitral incompetence resulting from absolute loss of leaflet substance, left ventricular chamber dilatation must occur to compensate for the volume regurgitated with each systole if cardiac output is to be maintained This in turn produces enlargement of the valve ring with relative

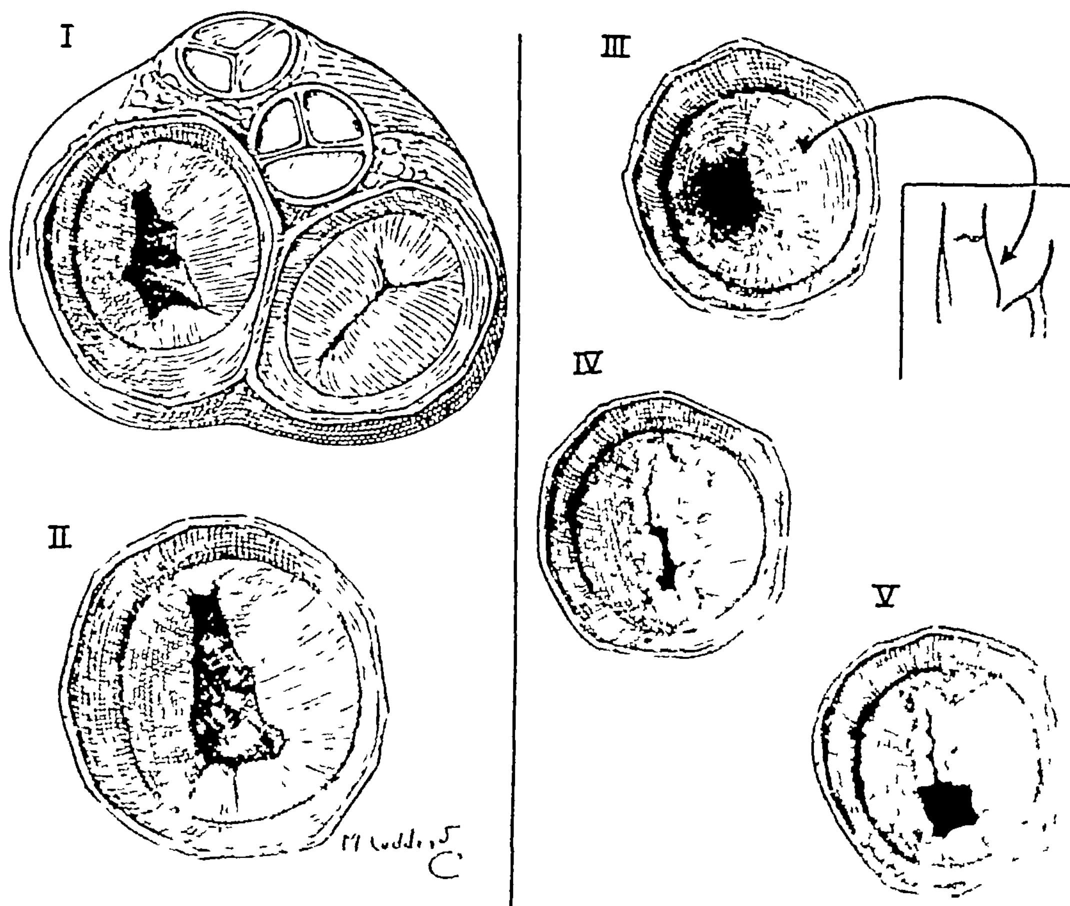


Fig 1 The five basic morphologic types of incompetent mitral valves I, Absolute loss of substance II, relative loss of substance, III, directional, IV, malocclusion, V, mixed pathology (From Harken et al. Journal of Thoracic Surgery, vol 28, Dec 1954)

loss of substance augmenting the regurgitation. Thus the peculiarly self-aggravating nature of this disease becomes apparent.

In addition to these "pure forms" of mitral regurgitation, there are three other forms in which the predominant problem is usually stenosis. In general, our policy has been to treat only the stenosis. Since there is some incompetence of the mitral closing mechanism, however, these pathologic processes must be included in a morphologic classification of mitral insufficiency.

TYPE III. DIRECTIONAL. This form of mitral insufficiency occurs when a stenotic mitral orifice faces into the left ventricular outflow tract (Fig. 1) The nature of the abnormally rigid stenotic orifice prevents closure, and its

position precludes obstruction by the ventricular wall in systole. This type of valve has been described previously as Group B mitral stenosis

TYPE IV MALOCCLUSION The regurgitation in this instance results from the inability of irregularly calcified leaflet margins to seat properly when the valve closes (Fig. 1) The result is a ribbon-shaped jet. Again, since mitral stenosis is the predominant lesion in most of these cases, correction aims only at relief of the obstruction.

TYPE V COMBINED PATHOLOGY Finally, a significant number of cases have been encountered in which fusion of leaflet margins and heavy calcification (the pathology common in pure mitral stenosis) will be found at the anterior commissure, while posteromedially there is a loss of leaflet substance without calcification, resulting in a significant area of insufficiency (Fig 1)

The quantitation of stenosis and insufficiency in such instances, and thus the management of such a lesion, presents a difficult problem. We have had numerous brilliant clinical results following the correction of stenosis alone when this type was found, even though there is substantial associated incompetence. When the degree of stenosis is negligible, however, treatment must be directed at the dominant insufficiency A discussion of how to determine whether stenosis or regurgitation is dominant in such a situation properly falls within the province of kinetic pathology or hemodynamics, but in order to emphasize the correlation of the morbid pattern with the dynamic valve function, it may be said that the identification of the significant lesion can often be made at the operating table. This is done by using the intra-atrial finger as a "baffle" or substitute for the lost leaflet substance, the regurgitant jet can thus be obstructed without interfering with leaflet mobility Ventricular filling proceeds normally as the leaflets fall away from the finger, but the leaflets plus the finger prevent regurgitation in systole. If the leak is significant, the blood pressure will rise from 20 to 50 mm. Hg within a few beats

Because the last three forms may enjoy remarkable improvement from the correction of stenosis alone, they must be excluded from the study

Now, let us turn our consideration to morphologic Types I and II We can add a third dimension to the pattern presented here. Normally, the annulus lies a centimeter, more or less, below the ventricular rim—*it is inside the ventricle*, if you will. This has important bearing on valve closure as Dr Hurwitt's movies have shown.

When a regurgitant jet occurs, it is near the postero-inferior aspect of the atrioventricular groove that the jet impinges on the left atrial wall. This part of the atrium dilates first. This part of the atrium is seen in roentgenograms and at the operating table to migrate *backward and downward* quite unlike the higher and more moderate dilatation of the left atrium in mitral stenosis Furthermore, autopsy examination of this zone reveals upward and backward migration of the annulus A sliding hernia of the valve complex has occurred. This shift and dilatation further shortens the posterior commissural leaflet and increases the incompetence. This has bearing on future corrective techniques In fact, for some three years now we have tried to reduce this herniation digitally by pressure on the atrium and attempted fixation of this position with a roll of Gelfoam and other extrinsic baffles How effective this has

been I can't say except that it reduced the jet at the term of operation. It was only used in mild cases.

HEMODYNAMICS OF MITRAL INSUFFICIENCY (KINETIC PATHOLOGY)

There are at least four features that should be reviewed.

1. The first of these involves the pressure relationships among the left atrium, the left ventricle and the aorta. It is at once apparent that the gradient in systole between the left ventricle and the left atrium is much greater than the gradient between the left ventricle and aorta. Thus, if the orifices were of similar size, the incompetent mitral opening would allow much more blood to regurgitate through it than would be ejected into the aorta as effective cardiac output. Nevertheless, in the face of this disadvantageous pressure relationship favoring loss of effective systolic ejection from the left ventricle, a quantity of blood equal to the combined aortic output and the regurgitant jet must flow from the atrium into the ventricle in each diastolic period That is, atrioventricular flow in diastole must equal the combined volume of blood put out into the aorta and back into the atrium during each contraction. In addition, there is another deterrent to maintaining adequate ventricular filling in diastole. The forward flow in diastole is at a low pressure gradient and therefore at a low velocity, whereas the reflux in systole is at a high gradient and velocity.

There are two principal mechanisms of compensating for this unfavorable dynamic situation. One is that the slower flow from atrium to ventricle may exist over a longer period of time, i e., *diastole may be prolonged.* The limitations of bradycardia are apparent although a given degree of incompetence will obviously be better tolerated with a slow rate. The principal mechanism by which circulation is maintained is therefore through change in the size of the mitral orifice so that the large volume of blood traveling at a low velocity in diastole proceeds through a larger orifice than the regurgitant jet in systole This depends on the maintenance of leaflet mobility (Fig. 2). The partial occlusion of the orifice by the ventricle or by annulus contractility is probably an additional minor factor. The cornerstone of our efforts at surgical correction of this lesion has been this important preservation of leaflet mobility so that the orifice can be larger in diastole and can become smaller in systole.

2. The second important concept of the kinetic pathology of this condition is that mitral regurgitation is a peculiarly self-aggravating disease When a significant amount of regurgitation occurs, a certain percentage of the left ventricular stroke volume is dissipated in retrograde flow. In order to maintain an adequate cardiac output, the left ventricle must dilate to accept a compensating volume of blood. This leads to an increase in the size of the annulus which in turn increases the *relative insufficiency* of the leaflets. More regurgitation leads to more dilatation, and a vicious cycle is established Thus, the self-aggravating nature of this condition is defined. Conversely, any reduction of reflux reduces ventricular volume, and as the mitral annulus shrinks simultaneously, the leaflets more nearly approximate Effective partial cor-

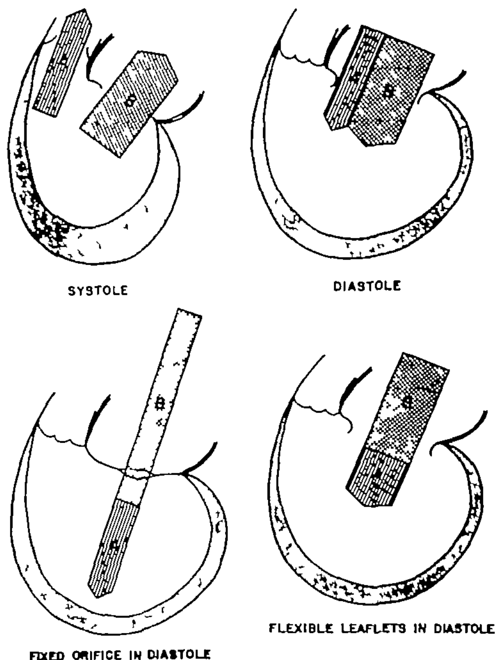


Fig 2. Upper pair· The combined volume of A and B must flow into the ventricle during each diastolic period. The pressure gradients are unfavorable to the forward flow as opposed to the rapid systolic reflux. Lower pair· The two principal compensating factors for this larger volume of forward flow at a lower velocity are prolongation of diastole and increase in the size of the mitral orifice (largely through leaflet mobility) (From Harken et al. Journal of Thoracic Surgery vol. 28, Dec. 1954)

rection of this lesion should therefore be *self-perpetuating*, provided that the ventricular myocardium has not exceeded the limits of its recuperative powers Failure to make adequate allowance for this dynamic change may result in the creation of stenosis by a procedure which attempts to correct the leak totally at the time of operation. Our experience in this regard will be described.

3 The third cardinal concept has already been mentioned. When the surgeon encounters free or significant mitral insufficiency on exploring the mitral valve, it is possible to hold the finger in the regurgitant jet at the leaflet level

in such a way that the leaflets slap firmly against it, and a more competent valve results. This is uniformly associated with a prompt elevation of the blood pressure by as much as 50 mm. Hg. Although instantaneous cardiac output determinations are not available, this prompt blood pressure elevation must correlate with an elevated cardiac output The reduction of these salutary changes can be simulated on a permanent basis by the surgical insertion of baffles.

4. Fourth, the regurgitant jet can be reduced by external pressure in the posterolateral aspect of the atrioventricular groove. This can be produced by either or both of two factors, namely annulus distortion or reduction of the sliding hernia of the valve complex. Either of these renders the leaflets more competent.



Fig 3 Autopsy specimen Ball baffle appears to lie in effective position beneath ventricular leaflet on left side of illustration Note Type I and Type II pathology Tricuspid valve is at right (From Harken et al Journal of Thoracic Surgery, vol 28, Dec 1954)

EVOLUTION OF BAFFLE PROCEDURES

In view of our inability to get grafts to live and to remain the same size as when inserted, we decided to use an inert Lucite ball baffle designed to be moved by the motion of the myocardium. This was the *ball baffle* operation This baffle was well tolerated in animals without embolus, and when placed in humans appeared even at autopsy to have been placed correctly and looked as though it would have functioned as designed (Fig 3). The explanation of its failure to obtain the proper hemodynamic result lay in the fact that in life, the chamber of the ventricle is more nearly spherical and the ball had been placed away from an effective position in the valve orifice To correct this a *bottle baffle* was designed to lie across the valve orifice (Fig. 4).

Of 23 patients operated on in 1952 and early 1953, only 6 are still alive The position of the bottle baffle was inconstant, so that late death from

unaltered decompensation or embolus or both rendered the operation unsatisfactory

Therefore, a technique was evolved for placing a *spindle baffle* through the mitral orifice (Fig 5), so that leaflet mobility was preserved, position was maintained and exposed suture and baffle ends were concealed to reduce the

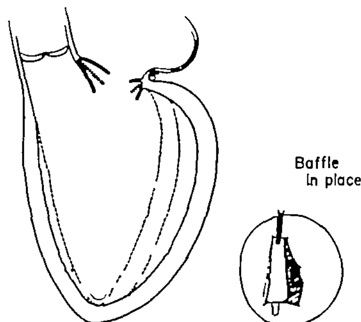


Fig 4 The bottle baffle lay in a more favorable position for obstruction of the regurgitant jet and preserved leaflet mobility Note bulging left ventricular myocardium and change in orifice size if leaflets are flexible. (From Harken et al. Journal of Thoracic Surgery vol. 28 Dec. 1954)

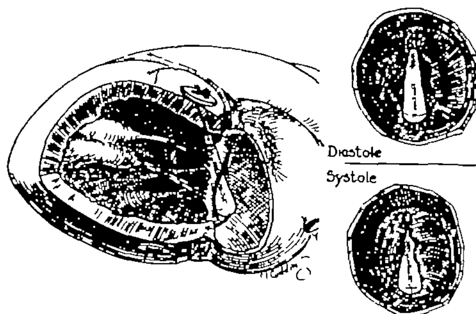


Fig 5 Technique of the spindle baffle operation. Cut-away detail of the position of the spindle baffle Note importance of leaflet mobility in allowing ventricular filling. (From Harken et al. Journal of Thoracic Surgery vol. 28 Dec. 1954)

danger of embolization. The position tended to correct valve herniation. That this baffle could be anchored as intended was attested when there was opportunity to observe the baffle at autopsy (Fig. 6).

Twenty-nine such Lucite spindles were placed in 1953 and 1954. There were 9 operative deaths, 10 late deaths and 10 are still alive. In one half of the late deaths there have been peripheral emboli, presumably from the baffle or its mooring. Two of the deaths (one operative and one late) were due to migration of the baffle. The other deaths (operative and late) must be



Fig 6 Autopsy specimen The spindle baffle is placed obliquely through the incompetent orifice and anchored firmly anterolaterally and posteromedially The narrow anterior end is below, and the wider posterior end above, the annulus (From Harken et al Journal of Thoracic Surgery, vol 28, Dec 1954)

ascribed to inadequate correction of the regurgitation or irreversible myocardial incompetence Otherwise, the operation would have been better tolerated and late deaths in congestive failure would not have occurred.

It can be argued that the patients, of whom this roentgenogram (Fig. 7) is representative, were operated upon in a phase of their life cycle when no local valvular corrective means could overcome the already irreversibly damaged myocardium and liver. This is not a valid argument for operation in a more favorable state. first, because some of these patients have had remarkable rehabilitation (as a matter of fact this patient (Fig. 7) is working as a secretary) and secondly, because the incidence of emboli with this operation precludes the use of this procedure in patients in whom it would be most effective



Fig 7 Roentgenogram of a patient in the spindle baffle series The left atrium comprises the bulk of the cardiac shadow extending to the left and right. Left ventricular enlargement is apparent in oblique views. Lung fields are relatively clear (From Harken et al. Journal of Thoracic Surgery, vol. 28, Dec 1954)

CONCLUSIONS

With these principles and this experience, efforts in the laboratory and on cadavers are being directed at the reduction of the size of the annulus and the downward shift of the herniated valve complex by direct suture. For more than a year we have worked on a principle of a double row of external reefing sutures stimulated by suggestion from Dr Paul C. Samson. Also, Drs William Bernhardt, Hugh E. Wilson and Richard Cardozo have been trying to effect these restorative steps in the open heart via the left atrium. This has been done in hypothermic animals with balloon obstruction of the ventricular outflow tract. During circulatory arrest the coronary and cerebral circulation have been perfused with oxygenated blood.

It is to be hoped that this experience will be more favorable and that a safe operation can be developed. Then patients can be accepted who can enjoy true rehabilitation.

METHODS OF SURGICAL TREATMENT FOR VALVULAR INSUFFICIENCY OF THE HEART

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PATHOPHYSIOLOGY

Practically all spontaneously developing valvular insufficiency may be considered to be the result either of a dilatation of the specific valve ring (annulus fibrosus) so that even completely normal leaflets become incapable of restraining leakage through the enlarged passageway (annular incompetence), or of shrinkage and retraction of one or more of the cusps so that they become unable to make a blood-tight edge contact with their fellows (organic incompetence) In other words, in both major types of regurgitation there is always either a relative or an actual dearth of valvular substance sufficient to provide valve competence.

Annular insufficiency apparently may be reversible in certain instances. Then it may be referred to as functional Also it may become progressively worsened. Compensation for insufficiency can be accomplished only by hypertrophy and dilatation of the appropriate ventricle which permits an increase in its gross output Then the net output, after loss by leakage, may still be adequate for all ordinary bodily needs and activities However, the annulus, especially if it already has exhibited the weakness which is implied by the term annular type of insufficiency, may well become further dilated as a consequence of this compensatory ventricular dilatation. This may result in an even greater degree of insufficiency which leads to an even greater ventricular dilatation in a further attempt to provide compensation. This vicious mechanism tends to persist, producing an ever increasing degree of regurgitation which inevitably must lead to myocardial exhaustion Conversely, anything which tends to break the vicious cycle may lead to at least a partial reversal of the entire process. This may occur naturally if developing stenosis gradually reduces the amount of regurgitation which can take place through the diminishing valve aperture Also its progress may be reversed to some extent by any surgical measure which lessens the insufficiency, thus permitting reduction in ventricular size and a concomitant decrease in the size of the annular ring

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An additional increment of annular insufficiency may become superimposed upon certain types of organic incompetence when the existing pathology in the valve or annulus permits subsequent "stretching out" of both the orifice and the annulus A classic example of this is the progressive worsening of the initially observed incompetence following inadvertent partial surgical interruption of the continuity of a valve leaflet or detachment of a significant portion of its natural supports during an attempt at the relief of mitral stenosis

ETIOLOGIC-PATHOLOGIC RELATIONSHIPS

ACQUIRED TRICUSPID REGURGITATION Acquired tricuspid regurgitation usually is caused by rheumatic valvulitis which, affecting the cusps, may produce shrinkage and deformation, or affecting the annulus, tends to produce weakening and dilatation of the ring The former condition necessarily is associated with an element of anatomic stenosis, which may be insignificant physiologically, may be equal in its effect to the insufficiency, or may be the preponderant element in the valvular malfunction. However, the latter type of regurgitation (due to annular dilatation), while commonly of rheumatic origin, also may be the result of any process which produces great right ventricular enlargement (dilatation) This may take place because the origins of the spiral musculature of the ventricle from the atrioventricular annuli fibrosi lend a sphincteric action and tonic support to the latter during systole.[1 2] In the presence of extreme ventricular dilatation, this support is reduced or lacking during the very important period of ventricular contraction. Hence, if there is any intrinsic congenital or acquired weakness of the annulus, the incessantly repeated surge of high intraventricular pressure during systole may be capable of initiating a degree of annular overstretching which ultimately will produce failure of valvular coaptation even in the presence of normal cusps Naturally, if any process associated with shrinkage or retraction of the cusps should be present, incompetence will be brought about more readily Once a physiologically significant degree of regurgitation has developed, compensation for it requires further ventricular enlargement. This, in turn, tends to cause further overstretch of the annulus fibrosus with consequent aggravation of the insufficiency And so, the vicious cycle continues

This phenomenon develops along similar lines with respect to both ventricles and their inlet (atrioventricular) valves, with one important clinical difference. That is—right ventricular function apparently is not essential to the maintenance of life[3 4 5] while at rest and in the presence of normal pulmonary vascular resistance. Increased systemic venous pressures which may rise to a level of 300 mm. water if gradually developed are tolerable (with reservations) to the mammalian organism and are sufficient to perfuse the pulmonary vascular bed adequately On the other hand, the effective pumping action of the left ventricle is the *sine qua non* of continued existence. even moderate mitral incompetence necessitates great limitation of activity, and severe mitral insufficiency leads to progressively increasing heart failure and early death.

ACQUIRED PULMONIC INSUFFICIENCY. Acquired pulmonary valvular competence nearly always is the result either of a dilatation of the pulmon annulus fibrosus or of surgical division of a congenitally stenosed valve. T former condition may develop in response to pulmonary arterial hypertens and the resulting presence of a greatly dilated right ventricle.

Although the origins of the spiral ventricular musculature from the arte annuli fibrosi may suggest a degree of tonic support to these valve rin actually the relaxation of the ventricular muscle fibers during diastole, period of maximal semilunar valve stress, would seem to imply a (norm lack of such support at the very time when it would be most useful. Besid

TABLE 1. CLASSIFICATION OF TYPES OF MITRAL INSUFFICIEN RECOGNIZED BY INTRACARDIAC EXAMINATION

	Number of Cases	*Percentage*
Annular dilatation	68	11 4
Organic insufficiency	193	32 4
Traumatic insufficiency		
Insufficiency created	243	40 9
Insufficiency increased	83	14
Congenital insufficiency	8	1 3
	595	100 0

TABLE 2 CLASSIFICATION OF CASES DEFINITIVELY OPERATEI ON FOR MITRAL INSUFFICIENCY

	Number of Cases	*Percentage*
Annular dilatation	52	31.3
Organic insufficiency	105	63 3
Traumatic insufficiency		
Insufficiency created	3	1 8
Insufficiency increased	6	3 6
	166	100 0

these rings would appear anatomically to be too incompletely or inadequatel supported by the ventricular musculature for this factor to be of prim importance in preventing such a tendency toward dilatation Probably we must indict a primarily weak ring structure in association with a pulmonar hypertension and a large right ventricular outflow tract (dilatation)

Since rheumatic disease rarely involves the pulmonary valve, sclerotic or retracting distortion of the leaflets and, hence, the coexistence of an acquired stenotic process are rare

ACQUIRED MITRAL REGURGITATION. Mitral valvular incompetence, as mentioned previously, is of the greatest clinical importance because the effective function of the left ventricle depends primarily upon the integrity

of function of its inlet and outlet valves (mitral and aortic) Ordinarily, mitral regurgitation is caused by the effect of rheumatic valvulitis, on the one hand directly deforming the mitral leaflets (73 9 per cent of our spontaneously developing cases were of this type), or, on the other hand, weakening the left

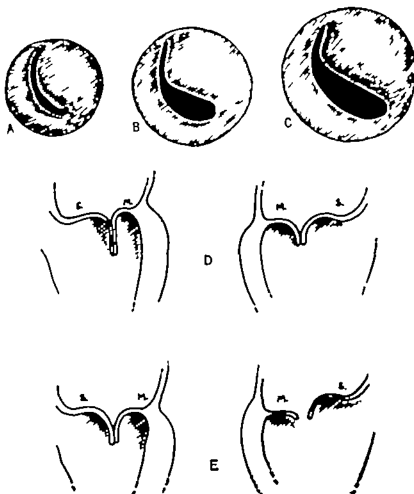


Fig 1 *A* Diagrammatic expression of normal mitral valve orifice. *B* Functional incompetence due to annular dilatation without intrinsic leaflet destruction. Note that the regurgitation takes place only in the posterior portion of the valve. *C*, Greater annular dilatation causes greater incompetence, and the 'aperture of incompetence becomes extended anteriorly (*A* *B*, and *C* from Bailey Surgery of the Heart. Courtesy of Lea & Febiger) *D* Diagrammatic illustration indicating that normally there is a greater systolic overlap of the mitral valve leaflets in the anterior portion of the valve (left drawing) than there is near the posterior commissure (right drawing) *E* With symmetrical dilatation of the mitral annulus, the anterior portions of the leaflets remain capable of making at least edge contact during systole (left drawing) long after the limited reserve of valvular tissue has been "used up" in the region of the posterior commissure (right drawing) rendering coaptation of the edges impossible and posterior regurgitation a reality

atrioventricular annulus fibrosus (26 1 per cent of our cases) (Tables 1 and 2) In the former condition, an associated element of mitral stenosis invariably is present. This stenosis may be clinically insignificant, it may be dynamically of equal importance to the insufficiency, or it may play the preponderant role in the over-all valvular malfunction. Not infrequently, both deformation of the cusps and dilatation of the annulus fibrosus may coexist. Presumably, in

many instances the latter condition has become superimposed upon the former.

It is notable that in mitral insufficiency due to annular dilatation, the leak always seems to take place in the posterior half of the valve orifice, usually being maximal in the region adjacent to the posterior commissure The anatomic basis for this preferential localization of the incompetence has been discussed previously.[6,7,8]



Fig 2 *A*, Severe (fatal) mitral regurgitation produced inadvertently during an attempt at mitral commissurotomy for stenosis Note that the septal mitral leaflet has been detached from all of its chordopapillary support in the region of the posterior commissure *B*, Diagrammatic illustration of condition produced by inadvertent surgical division of the continuity of the mural leaflet *C*, Valve repair accomplished by the use of a pericardial suture and the "mitral stitcher," a special mechanical suturing instrument

Serious mitral regurgitation resulting from inexpert attempts at mitral commissurotomy usually takes the form either of interruption of the continuity of one of the cusps, or of detachment of one of them from a major portion of its chordopapillary support (Fig. 2). Once this initial severe leak has been created, Nature's attempt at compensation, involving dilatation and hypertrophy of the ventricle, leads to progressive dilatation of the left atrioventricular ring, thereby bringing about a worsening of the leak.

The most common type of mitral insufficiency, of course, is that due to deformation of one or both leaflets, either from shrinkage of the valve substance or retraction of the free margin toward the ventricular wall (Fig 3

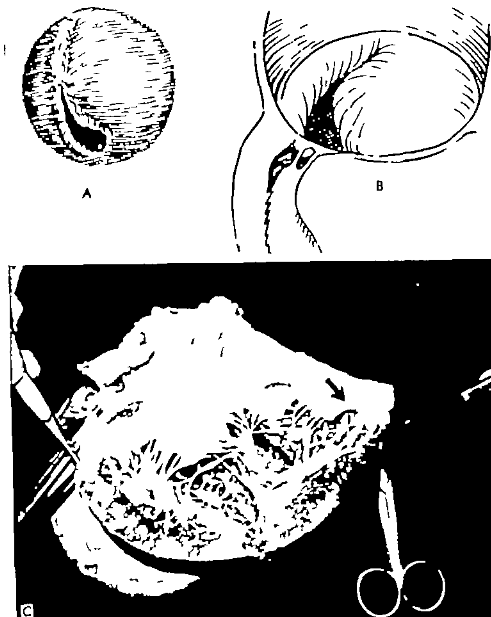


Fig 3 *A* Posterior "teardrop insufficiency of the mitral valve due to shrinkage and deformation of its leaflets, usually the mural. Note apparent loss of mural leaflet substance posteriorly There is nearly always in these valves an element of coexisting stenosis due to anterior cross fusion of the leaflets (commissural obliteration) *B* Regurgitation due to mechanical retraction of posterior commissural valve tissue toward the ventricular wall Usually this is brought about because of primary lateral fusion of the posterior papillary muscle to the ventricular wall. Thus, it tends to draw the attached portion of the valve toward the ventricle *C* Incisura of incompetence" due to intrinsic shrinkage or loss of leaflet substance shown in a human mitral valve which happens to be multicuspid. (Bailey· Surgery of the Heart, courtesy of Lea & Febiger)

A, *B*, *C*) Not infrequently, a serious degree of associated mitral stenosis renders it necessary to open the obliterated anterior commissure in a patient with posterior retraction of the mural leaflet (teardrop valve) Commissurotomy then, necessarily, is limited to the anterior commissure in this type of case (Fig. 3*A*) When commissurotomy is indicated for the associated stenosis in the arcuate type of valve, both commissures should be opened. Often the regurgitant element may become reduced significantly after appropriate com-

missural separation because each of the anatomically mobilized leaflets may become better able to adjust to the other's marginal outline.

ACQUIRED AORTIC INSUFFICIENCY. Since competence of the aortic valve has approximately equal importance in ventricular function to competence of the mitral valve, a large degree of aortic incompetence leads early to great ventricular overwork and ultimately to failure and death. Although the patient, relatively speaking, may be able to maintain compensation for a long while, once clinical symptoms begin the course is rapidly downward.

The signs and symptoms associated with aortic regurgitation may be divided into two groups: (1) those which are due to myocardial exhaustion and failure from overwork, and (2) those which are due to coronary insuffi-



Fig 4 *A*, Preponderant calcific aortic stenosis with an associated element of less important aortic insufficiency (Bailey Surgery of the Heart, courtesy of Lea & Febiger) *B*, Dynamically preponderant aortic regurgitation in a valve presenting the classical pathology of rheumatic (calcific) aortic stenosis

ciency which is relative in rheumatic cases (due to increased myocardial nutritional demands in the presence of an impaired mechanism of coronary filling), but may be intrinsic (due to narrowing of the coronary ostia) in luetic ones.

As in involvement of the atrioventricular valves, the rheumatic process may deform the cusps of the aortic valve with resulting loss of substance, or it may weaken the structural components (elastic and collagenous fibers) of the aortic annulus fibrosus so that dilatation occurs, rendering even normally developed cusps incapable of providing competence to the greatly enlarged arterial channel.

While annular aortic insufficiency due to syphilis now has become rather rare in the United States, that form due to the rheumatic state still is rather common. This condition statistically has made up only 25 per cent of our patients who have presented themselves with clinically significant aortic regurgitation However, it has made up almost 50 per cent of those who have

been operated on definitively for this lesion, mainly because these patients present a more severe, a more "wide-open" type of insufficiency with a diastolic pressure usually determined at a level close to zero (see Figs 11, 12 and 13)

Rheumatic regurgitant intrinsic cusp deformation follows the same pattern that is observed in rheumatic aortic stenosis Necessarily, it is always associated with an element of the latter Indeed, in a majority of cases with detectable combined stenosis and insufficiency the former is clinically the dominant lesion, the amount of leakage being small and of little dynamic significance.

ACQUIRED INCOMPETENCE OF THE ATRIOVENTRICULAR VALVES

While it would seem logical to treat valvular insufficiency due to annular dilatation by tightening a constrictive band or sash placed about the appropriate annulus fibrosus, and while this procedure has been attempted, it has not yet become a practical reality with respect to either the mitral or tricuspid valve.

Obviously, constriction of an atrioventricular annulus would be of little value in that form of regurgitation which is due primarily to leaflet shrinkage and retraction, unless an element of functional insufficiency had become superimposed upon the organic. Then, of course, the maximum beneficial effect which one could hope to obtain would be but relief of that increment of the incompetence which is due to the annular dilatation.

In our own laboratory, a simple surgical method of direct approach to the mural portion of either atrioventricular valve annulus fibrosus has been worked out. We have felt that such a technique, while reducing the annular circumference less than an encircling technique, would be much safer It consists simply of dissecting the fat pad from the respective atrioventricular groove along with the circumflex coronary artery and any associated veins (coronary sinus on the left), and displacement of these structures downward (toward the respective ventricular apex) (Fig. 5*A*, *B*, Fig 6*A*, *B*) In one recently operated patient with moderately severe annular incompetence of the tricuspid valve, four such plicating sutures resulted in complete abolition of the previously noted regurgitant jet to the satisfaction of each of the four members of the operating team after repeated intracardiac palpatory explorations Unfortunately, preoperative cardiac catheterization studies had not been carried out in this individual.

Already several methods have been tried clinically in the treatment of organic mitral regurgitation,[6 9 10 11 12 13 14] and several others have been used experimentally [15 16] Each of these methods has presented some virtues and some disadvantages The chief disadvantage of the method which embraces placement of a nonmoving pedicled transventricular subvalvular pericardial diaphragm[5 8] is that in some cases it may not lie in close enough approximation to the ventricular aspect of the mitral orifice completely to prevent the regurgitant leak (Fig 7*A*, *B*, *C*, *D*)

Consideration of the manner in which such a diaphragm must work led inevitably to certain previously stated basic conclusions [5 8] The diaphragm represents an attempt to implement the concept of replacement of an actual or a relative loss of valvular substance with a graft of living tissue. The effect

and unusually terminal mitral regurgitation which presents an area of leak larger than one square centimeter[17] Hence the terminal phalanx of the index finger (1 4 sq cm. cross section) would seem usually to be the largest cone or plug which would be required to block such a leak. It was inevitable that idle thoughts should arise of leaving the actual finger or a prosthetic facsimile extending through the valve permanently in order to maintain this effect.

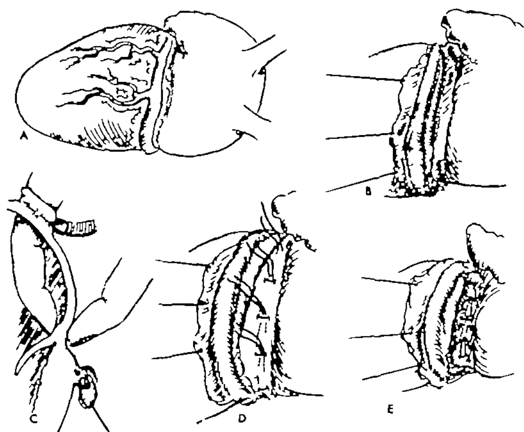


Fig. 6 *A* Exposure of coronary sinus and fat pad occupying the left coronary sulcus. *B* By dissecting the fat pad forward after dividing the epicardium over its atrial aspect the depths of the groove and the underlying mural portion of the left atrioventricular annulus fibrosus are exposed to direct examination and surgical repair Since the important tributaries of the circumflex branch of the left coronary artery all run toward the left ventricular apex they are unaffected by this mobilization. *C*, Bimanual examination of the annulus fibrosus is carried out by inserting the operator's ungloved index finger into the left atrium (via the appendage) *D* Guided by the intracardiac finger a series of interlocking mattress sutures (of heavy silk) are applied directly into the mural portion of the circumference of the fibrous ring *E* By tying down these mattress sutures, the size of the left atrioventricular annulus (and of the channel it surrounds) is reduced markedly The intracardiac finger can appreciate the change in valvular action and estimate the extent of diminution brought about in the previously recognized regurgitant jet.

The inferiority of prostheses to living tissue grafts in general, and especially in those areas of the body where movement is continuous, naturally led to consideration of various possible ways of using a living tissue graft for this purpose. It was essential (1) that it should be an autogenous graft (to obviate the onset of late degenerative changes), (2) that it should be largely flexible (to adjust the rhythmic changes in the size and shape of the heart), (3) that its cross sectional contour should be appropriate to, or adaptable to, the shape

of the "aperture of incompetence" (that portion of the valvular opening which is persistent during systole in an atrioventricular valve), (4) that it should not swell initially, and (5) that it should not shrink appreciably with the passage of time. Glenn[16] has already proposed the use of the mobilized pedicled internal mammary artery and vein with attached fatty tissue and covered with a free inverted vein graft, using a similar concept and similar specifications. However, Cleland[17] already had used pedicled pericardial tubular grafts passed through the incompetent portion of the valve orifice in 1953.

After considerable thought and animal experimentation, a graft consisting of an appropriately carved segment of costal cartilage incorporated within a

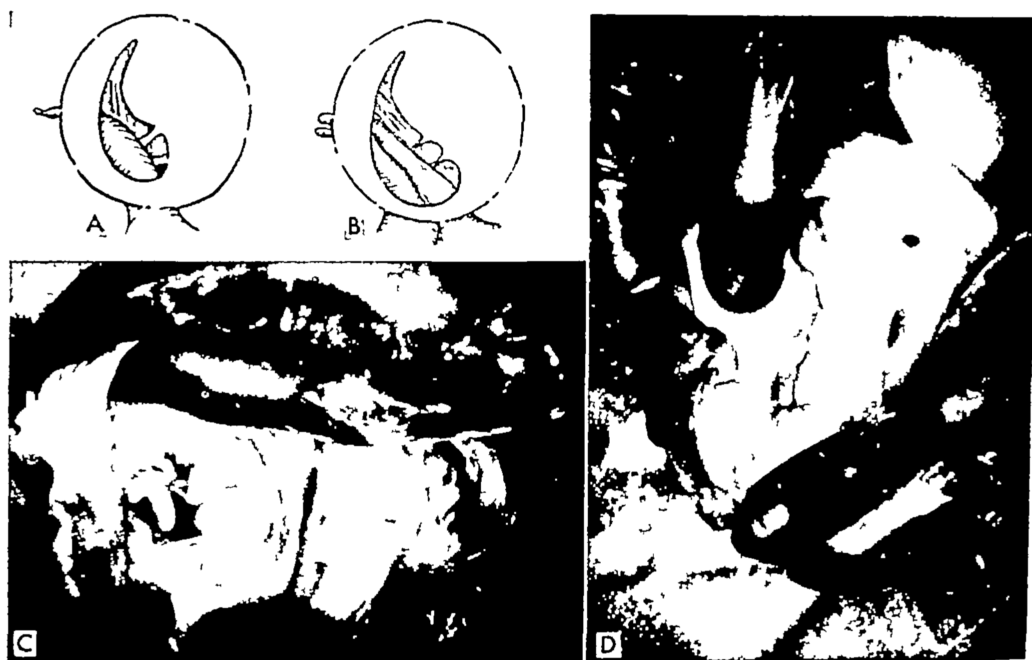


Fig 7 *A*, Incompetent mitral valve with posterior leakage treated by placement of subvalvular pedicled pericardial graft *B*, If the "aperture of incompetence" is too large for a single graft, a second pericardial tube may be placed alongside it (*A* and *B* from Bailey Surgery of the Heart, courtesy of Lea & Febiger) *C*, Photograph of pedicled pericardial graft which was placed across an incompetent mitral valve three years previously Diastolic position *D*, Same valve in systolic position, showing the very adequate tamponading effect of the graft

tightly fitting multiply perforated tube prepared from a long free graft of the patient's pericardium has been considered to fulfill these criteria satisfactorily. By carving the costal cartilage so that it is larger at one end than at the other, but smoothly tapering so that its cross-sectional contour is similar from one end to the other, provision is made for easy adjustment in the effective size of such a pericardio-cartilaginous "stent," by drawing it through the valve orifice to a greater or lesser extent The cross-sectional outline of the cartilaginous portion of the transventricular stent essentially is made to simulate that of the usual alar air foil for the so-called "teardrop" type of insufficiency (Fig 17*A*). This contour is equally suitable for the tamponade of annular mitral insufficiency since, in these cases, the "aperture of incompetence" assumes a location and configuration similar to those of the "teardrop" type

However, for the 'arcuate" form of insufficiency the cross-sectional contour of the stent must resemble that of the geometric figure described as a ' segment on a chord" of a circle (Fig 19*B*) The necessity for a wider and flatter stent in this latter type of insufficiency commends the use of a portion of the lower costal marginal cartilaginous complex for its preparation.

In the development of a technique suitable for the insertion and proper placement of such a transvalvular stent, it soon became evident that since the stent must be maintained in close contact with the posteromedial commissure, at least in the annular and in the "teardrop" types (after an appro-

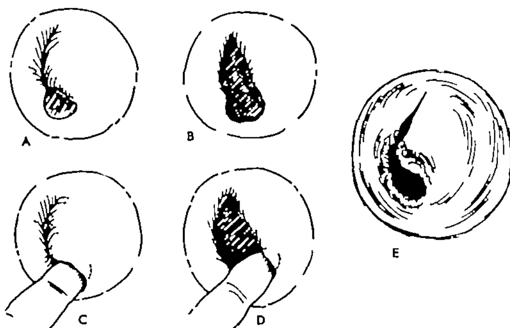


Fig. 8 *A* Posteriorly incompetent mitral valve in the systolic position. *B* Same valve in diastole. *C* Tamponade of aperture of incompetence' with finger tip inserted into incisura. Systole. *D* During diastole a very adequate valve aperture is present even with the tamponading finger in place. Note that mobility of the entire septal and of the anterior portion of the mural leaflet is unhampered *E*, In regurgitation associated with significant stenosis, it is essential that the latter first be overcome by an appropriate commissurotomy operation before a definitive attack upon the regurgitation be undertaken. (Bailey Surgery of the Heart, courtesy of Lea & Febiger)

priate anterior commissurotomy) of insufficiency, its upper site of attachment or suspension must be located somewhat to the right posterior aspect of the left atrium. This would often seem to imply the necessity for a right-sided thoracic approach in the treatment of mitral insufficiency However, by elevating the apex of the heart the entire posterior aspect of the left atrium may be exposed to view through a left-sided incision.

On the other hand, a suitable ventricular site of suspension which must be placed somewhat near the apex would seem to require a left-sided thoracic approach. The necessity for removal of a long and fairly wide strip of the patient's own pericardium in order to provide the supporting covering and terminal attachments of the stent also would urge a left-sided surgical approach. Therefore, the problem may be answered either by the usual long

left fifth intercostal incision, posterolaterally, or by a combined anterior approach to the heart through an incision made in the anterior right 3rd or 4th intercostal space, and also through another which opens the left 5th or 6th intercostal space anterolaterally. So far it has not seemed advisable to us to transect the sternum in any of these cases.

At the time of presentation of this manuscript, this procedure had been carried out in only 3 patients, all of whom had very severe (one, 3 plus; two, 4 plus) mitral insufficiency. Two of the incompetent valves were of the organic type (posterior teardrop), while the other one was considered to be of the annular type. The first patient (H. G.), a 40-year-old white female, was operated upon on February 23, 1955. All have survived In each the regurgitation has been reduced greatly to digital examination. In two, the preoperatively prominent systolic murmur scarcely can be heard postoperatively. In all, a clearly audible first sound, preoperatively absent or replaced by the murmur, has been restored. There is no suggestion that there has been creation of a clinically detectable element of mitral stenosis in any The method appears to be just as effective in controlling regurgitation of the annular type of insufficiency as it is with intrinsic organic deformity of the leaflets themselves

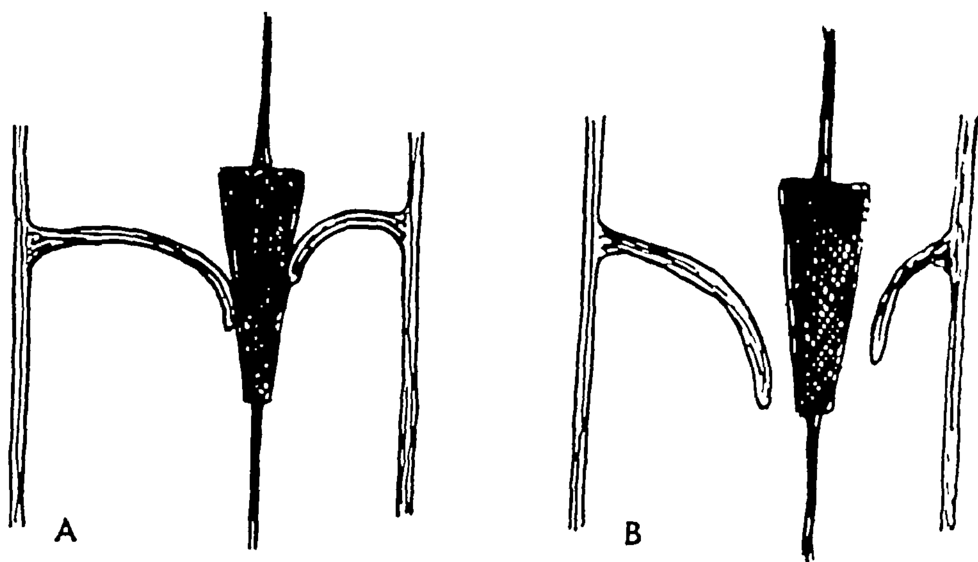


Fig 9 In valves with unequal levels of suspension of the leaflets so that their edges can not meet at the same apicobasal level, a transvalvular stent may provide them with a common central plane of contact, thus abolishing the leak *A*, Valve in systole *B*, Valve in diastole

In the arcuate form of insufficiency in which the free margin of one leaflet often lies at a higher level within the atrioventricular canal than does the other, the stent would seem to offer another important, although originally unanticipated, advantage Since a portion of it extends below the valve orifice, while the other end protrudes above it, the edges of the individual cusps in the region of the regurgitant leak may each make accurate systolic contact with the stent at different levels although they may be incapable of making mutual contact at any single given level, thus cutting off any element of leak which may be due to the unequal levels of valvular suspension (Fig. 9*A*, *B*).

THE HUFNAGEL VALVE

Hufnagel's[18 19 20] ingenious and efficient plastic ball valve of Lucite is discussed elsewhere in this book. For several reasons, it would seem that the treatment of aortic insufficiency by the use of the Hufnagel valve is unphysiologic in an over all sense. In no instance should it be applied in patients presenting clinical coronary insufficiency or a prominent element of coexisting aortic stenosis

TREATMENT OF ORGANIC AORTIC REGURGITATION WITH A TRANSVALVULAR STENT

In aortic insufficiency associated with rheumatic valvular leaflet deformation, there always is a coexisting element of aortic stenosis If it is dynamically significant, an aortic commissurotomy performed by way of the supravalvular approach should be carried out before undertaking definitive corrective surgery for the insufficiency Then, the palpating intra-aortic finger may evaluate the remaining "aperture of incompetence (valve opening maintained during diastole in a semilunar valve) and may determine its size and shape. It usually is a somewhat triangular aperture (see Fig 4*A*, *B*)

The operator then prepares a resected costal cartilage (usually the fourth or fifth), carving it to the desired cross-sectional contour, and causing it to taper so that it varies from an obviously excessive size at one end to an obviously inadequate one at the other end. This stent, like the one used for mitral insufficiency, is encased within a long perforated pericardial tube, which in this instance is made with two suspending tails at the broad end and a single heavier one at the smaller end. The stent is dragged through the opening in the aortic wall and into the valve, as indicated in the section on technique (see Fig 22*D*) The stent is drawn cautiously into the valve orifice until it seems just to occupy the entire 'aperture of incompetence." Then, the lower pericardial extension is affixed securely to the ventricular wall while the upper tails are caused separately to penetrate the aortic wall at such sites as seem suitable to prevent rotation of the stent out of appropriate alignment with the valvular angles Very definite favorable changes in the pulse and blood pressure curves were established by this means in the one patient treated prior to the time of this presentation.

ANNULAR DILATATION OF THE SEMILUNAR VALVES

Surgical constriction of dilated pulmonary and aortic annuli was carried out at the Hahnemann Hospital in Philadelphia, for the first time in selected patients, on January 29, 1954, and February 25, 1954, respectively The actual technique is extremely simple for the former condition. After the aorta and pulmonary arteries are anatomically dissected apart at their bases, a sash of nylon fabric is passed about the pulmonary conus and beneath the conus ligament. It is tightened until the shock of valvular closure becomes digitally palpable, and then further until the development of a systolic thrill (bruit) over the pulmonary artery indicates the beginning production of stenosis Then, the sash is tied and the procedure is terminated (Fig 10*A*, *B*, *C*)

The analogous procedure for the treatment of aortic insufficiency in the

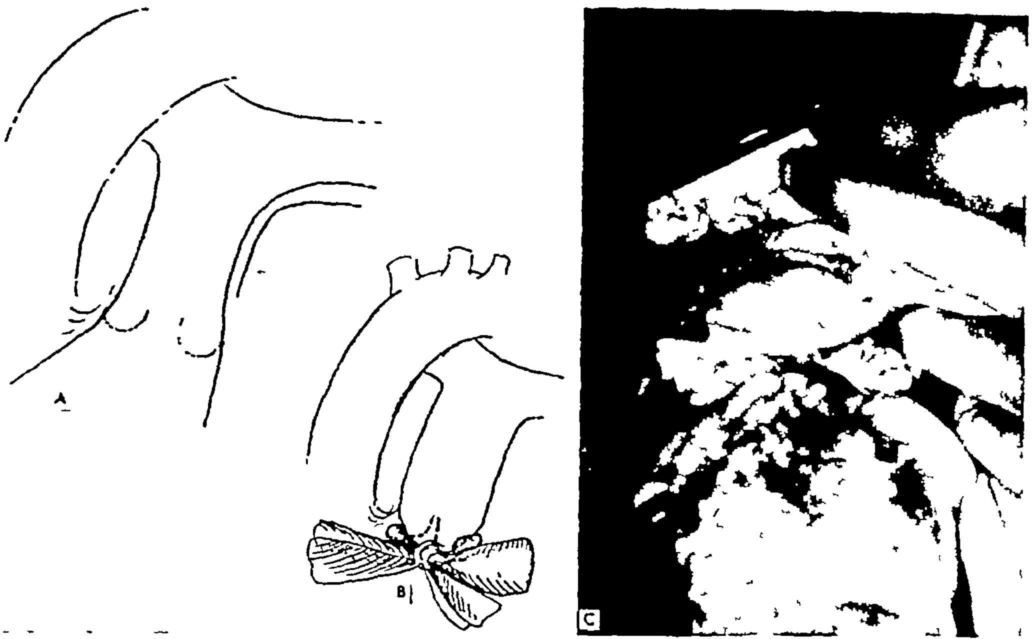


Fig 10 *A*, Diagrammatic representation of incompetence of pulmonary valve—annular dilatation *B*, Surgical constriction of pulmonary annulus fibrosus, reestablishing valve coaptation and restoring full competence *C*, Actual photograph taken immediately after constriction of an incompetent pulmonary valve with a nylon sash There was immediate disappearance of the diastolic thrill previously palpable over the anterior surface of the right ventricle. Simultaneously, the palpable shock of pulmonary valve closure was reestablished This corresponds to the auditory pulmonary second sound

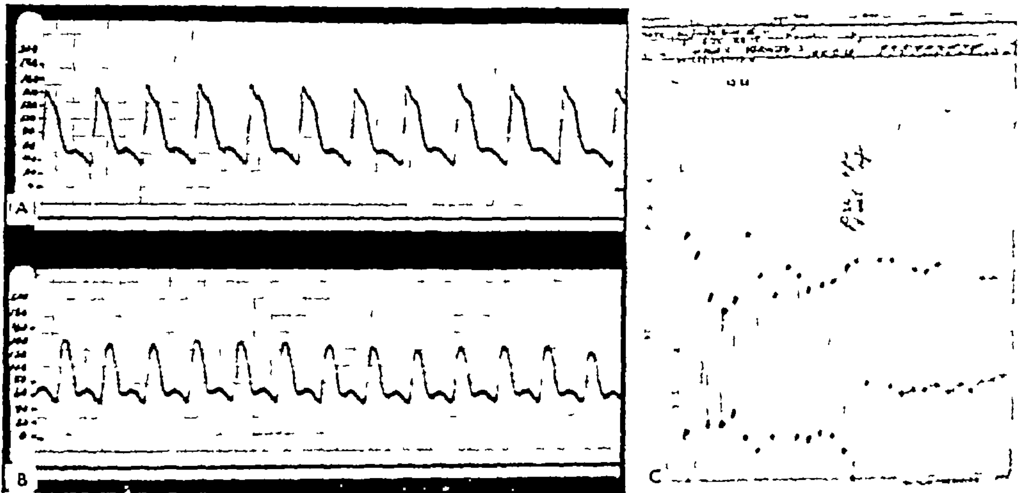


Fig 11 *A*, Brachial arterial pressure tracing taken during surgery in a patient with aortic insufficiency due to annular dilatation associated with luetic aortitis *B*, Immediate change in brachial arterial pressure tracing in this patient following tight wrapping of the dilated ascending aorta with nylon fabric strips and constriction of the aortic annulus with an encircling nylon fabric sash *C*, Chart of blood pressure determinations obtained in this patient before and after surgery

absence of associated stenosis is considerably more difficult and the technique required is complicated. However, it can be carried out without great operative risk by an experienced operator, and usually brings about a permanent and complete correction of the valvular incompetence with the consequent logically to be expected physiological and symptomatic benefits (see Figs. 11, 12 and 13).

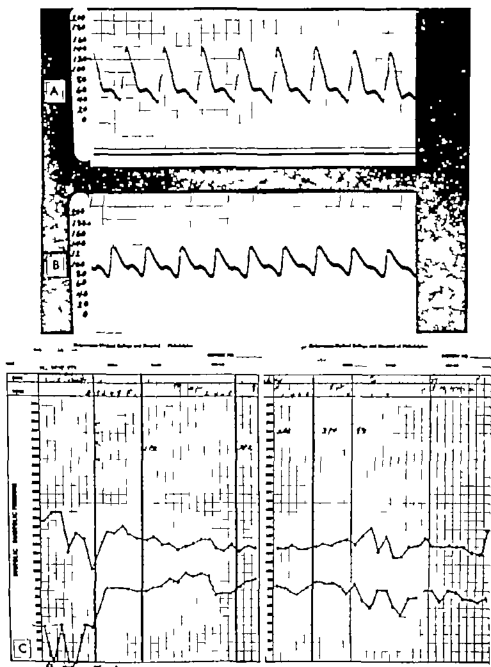


Fig. 12. *A* Brachial arterial tracing obtained at surgery in K. McG a 16-year-old white male with a history of previous rheumatic fever and a wide-open aortic insufficiency *B* Alteration in brachial pressure curve produced by tightening a constricting sash placed about the aortic annulus. Again disappearance of the diastolic thrill, appearance of the shock of cusp approximation and development of a systolic thrill over the aorta indicated overcorrection of the valvular leak. *C*, Chart of blood pressure determinations in K. McG showing postoperative elevation of originally very low diastolic blood pressure level.

exact location of the fibrous ring. Again, plicating mattress stitches of heavy silk were applied into the annular substance by direct suturing through the wall of the heart. The long axis of the sutures was placed parallel to that of the fibers of the annulus. Again, an overlapping technique of placement of the sutures was employed. By tying down all of these stitches, a remarkable reduction in the length of the mural portion of the annulus (and of the length of the mural leaflet) was brought about (Figs. 6*A*, *B*, *C*, *D*, *E*, *F*, and 15*A*, *B*, *C*).

However, as mentioned, the leak was not completely abolished in any of these clinical cases. The location of the "aperture of incompetence" in each patient seemed to become displaced forward, away from the posterior com-

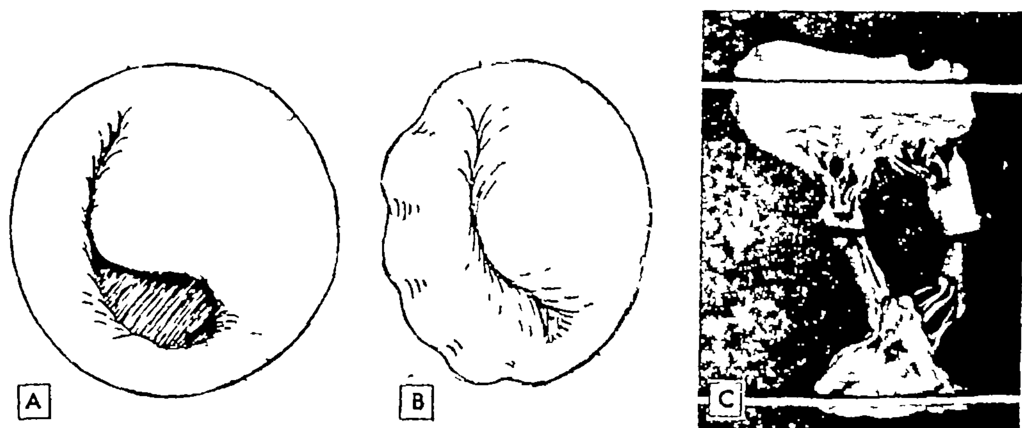


Fig 15. Reduction in the length of the mural portion of the mitral valve ring by plicating mattress sutures *A*, Annular incompetence of the mitral valve—leaflets in diastolic position Note posterior incompetence *B*, Marked reduction in incompetence due to plication of mural portion of annulus *C*, Photograph showing (normal) relative disparity in lengths of extracorporeal mural (top) and septal (bottom) mitral leaflets Reduction in the length of the mural leaflet tends to reduce this disparity which becomes exaggerated in annular incompetence to a more normal relationship, thus tending to restore competence (From Bailey Surgery of the Heart Courtesy of Lea & Febiger)

missure and into the middle of the valve orifice, as a result of this mural plication. Whether the sutures inadvertently brought about some distortion of the mural leaflet, or whether there was a coexisting unrecognized element of organic distortion due to intrinsic leaflet disease, is not known.

APPLICATION OF A TRANSVALVULAR SCULPTURED STENT FOR MITRAL INSUFFICIENCY. While previously unsuspected incompetence of the mitral valve may well be encountered for the first time during the performance of commissurotomy through a left-sided thoracic incision, we prefer whenever feasible to approach the problem electively from the right side.

With the patient in the supine position, a curved inframammary incision is made over the right anterior hemithorax. The pectoral muscles are split as far as possible in the line of skin incision, and hemostasis is secured carefully. The right fourth anterior intercostal space is opened widely, the internal mammary vessels being divided between ligatures The lung is compressed posteriorly and the pericardium is opened in "L" fashion, the horizontal arm of the incision running in a cephalad direction just anterior to and parallel

with the right phrenic nerve. The vertical arm begins at a level just caudal to the right auricular appendage and runs anteriorly to the sternal edge. The interatrial groove is dissected for a limited distance, 1-1 5 cm., and down to a depth of 1 cm. After encircling the area of dissection with a purse-string suture of heavy nylon suture material, the left (posterior) side of the partially separated interatrial septum is incised. The operator's ungloved left index finger is inserted through this stab wound into the left atrial chamber to explore the mitral valve. The magnitude, type and primary site of localization of the regurgitation are evaluated (Fig 16*A*, *B*, *C*)

If significant stenosis is found, a digitally performed or digitally guided instrumental commissurotomy should be carried out. Reexamination will disclose whether the regurgitation has been bettered or worsened by the leaflet mobilization, its approximate amount (in a range of 1 to 4 plus), and its site of localization within the valve orifice. If it is rated as either 3 to 4 plus,



Fig. 16 *A* Double anterior thoracic incision used in placing a transvalvular pericardio-cartilaginous stent for mitral insufficiency The right third or fourth and the left fifth intercostal spaces are opened. *B* Limited dissection of the interatrial groove in order to insert operating finger into the left atrium from the right thoracic approach. *C* Insertion of left index finger into left atrial chamber after making a stab incision into left half of split interatrial septum. Hemostasis is provided by an external purse string suture.

transvalvular application of a pericardio-cartilaginous stent is indicated. The finger is withdrawn from the heart and the dissected interatrial groove is constricted temporarily by applying tension to the encircling purse string

Now, a left anterolateral thoracic incision is created, opening the fifth intercostal space. The sternum is not transected. The lung is compressed posteriorly and a strip of pericardium 5 cm. in width, extending the full length of the sac anterior to the left phrenic nerve, is excised. If the aperture of incompetence presents a teardrop outline, the fifth costal cartilage is resected for preparation of the stent. If the valve is arcuate in type, a portion of the broader, flatter, costal cartilaginous marginal complex, of which the sixth is a major component, is removed

The cartilage, which should be about 3.5 cm. in length, is carefully carved in a manner resembling that used in soap sculpture. While it is made broader and thicker at one end, tapering to a smaller opposite end, it must maintain the same type of cross-sectional contour for its entire length. The broader end is designed to be the atrial extremity, while the smaller one is destined to lie within the ventricular chamber

All possible fat is removed from the resected (autogenous) pericardial patch and it is spread out on a moistened towel with the epicardial (smooth) side down. The carved cartilaginous mass is placed longitudinally upon it in a central position with its flat side downward. The pericardium is folded longitudinally over the mass, and the lateral excess is trimmed to fit snugly leaving only a 2 or 3 mm. overlap. Arterial silk (No. 4-0) sutures are used to transfix the mass, attaching it to both sides of the created pericardial tube. The tube is completed by approximating the cut pericardial edges with interrupted silk sutures. At least a dozen 4 mm. longitudinal perforations are made in both sides of the pericardial tube down to the cartilage to prevent internal fluid collection and consequent over-all swelling of the stent. The longitudinal excess of pericardium, at the larger extremity of the cartilaginous mass, is trimmed to provide a suspending tail attached only at its posterior margin (the thicker one in a stent carved to fit a teardrop valve), the region which is to be applied into the posteromedial portion of the valve orifice. The entire pericardial excess attached to the apical extremity of the mass is rolled longitudinally to form a very strong rope-like suspending tube or "tendon." Individual heavy sutures of nylon are affixed to the free ends of these apically and basally attached tails. When the stent is to be applied entirely from the left side, two tails may be prepared for the broader end, the apex being suspended by a single heavier one (Fig. 17*A*, *B*, *C*).

A purse-string suture is placed about a relatively avascular portion of the anterolateral surface of the left ventricle near the apex. The operator's ungloved left index finger is reinserted into the left atrial chamber via the dissected interatrial groove (from the right side)

With his right hand, the operator inserts a terminally threaded semi-malleable probe through the circumscribed area on the left ventricular wall (through the left thoracic incision). The intracardiac finger tip inserted momentarily through the incompetent valve recognizes the probe tip as it is advanced up the ventricular chamber, and guides it until it emerges from the dissected interatrial groove. An assistant picks up the suture loop borne in the terminal eye and the probe is withdrawn. Bleeding from the ventricular puncture may be restrained by tightening the purse string placed about it.

The suture in the apical "tail" of the stent is attached to one strand of the (divided) suture loop which emerges from the interatrial groove The other suture strand serves as a "spare" in case of graft detachment from the first one and is temporarily left in place, traversing the chambers of the left side of the heart. By traction upon the appropriate suture end emerging from the puncture wound in the ventricle, the apical pericardial tail is drawn into the left atrium by way of the interatrial groove, sliding upon the palmar surface of the index finger. Usually, the cartilaginous mass cannot enter the heart while the finger occupies the created passageway between the atria even after complete relaxation of the purse string However, as the finger momentarily is withdrawn from the opening, the cartilaginous portion of the stent is drawn quickly into the left atrium, the finger tip following it immediately to tamponade the septal incision Further traction causes the stent to enter the actual valve orifice. It tends to lie close to the posteromedial commissure

Alternate traction upon the two sutures attached to the tails of the graft soon establishes the exact level of placement (and hence the appropriate size of its applied cross section) which affords maximal reduction in the palpable regurgitant jet. The ventricular attachment of the emerging apical pericardial tail is made secure by tying the encircling purse string in the ventricular

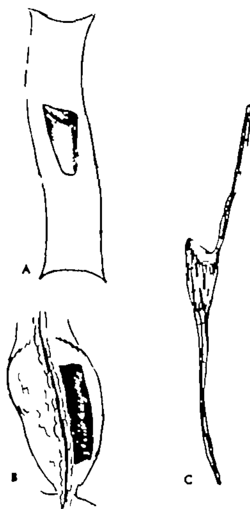


Fig. 17 *A* Sculptured tapering segment of costal cartilage is laid upon resected strip of pericardium. Usually, the cross section of the stent is made to present a contour similar to that of the aeronautical alar airfoil. *B* Defect left in pericardial sac by partial resection. *C*, Completed pericardio-cartilaginous stent. Multiple small longitudinally directed "piecrust" perforations in the pericardium reduce the risk of accumulation of fluid under the pericardial covering of the cartilage.

myocardium about it, and then penetrating the emerging end of the pericardial tube by the attached Atraumatic needle of the purse string and then retying The site of atrial fixation is rendered secure by removing the finger and penetrating the pericardial tail emerging from the dissected interatrial groove with the oversewing hemostatic stitches used to approximate its edges (Figs. 18*A*, *B*, *C*, and 19*A*, *B*, *C*, *D*)

The right side of the pericardial sac is repaired (to prevent subsequent possible right-sided dislocation of the heart) and meticulous attention is



Fig. 18 *A*, Terminally threaded probe is passed through purse-stringed avascular site near left ventricular apex Tip of probe is found by intracardiac finger and is guided to emerge from the right interatrial groove An assistant grasps the suture loop borne in the terminal eye *B*, After removing the probe from the heart, the loop is divided and one end is attached to the free extremity of apical "tail" of the stent. *C*, By traction upon the ventricular end of the appropriate suture, the cartilaginous mass is drawn into the left atrial chamber as the intracardiac finger is removed momentarily from the interatrial groove

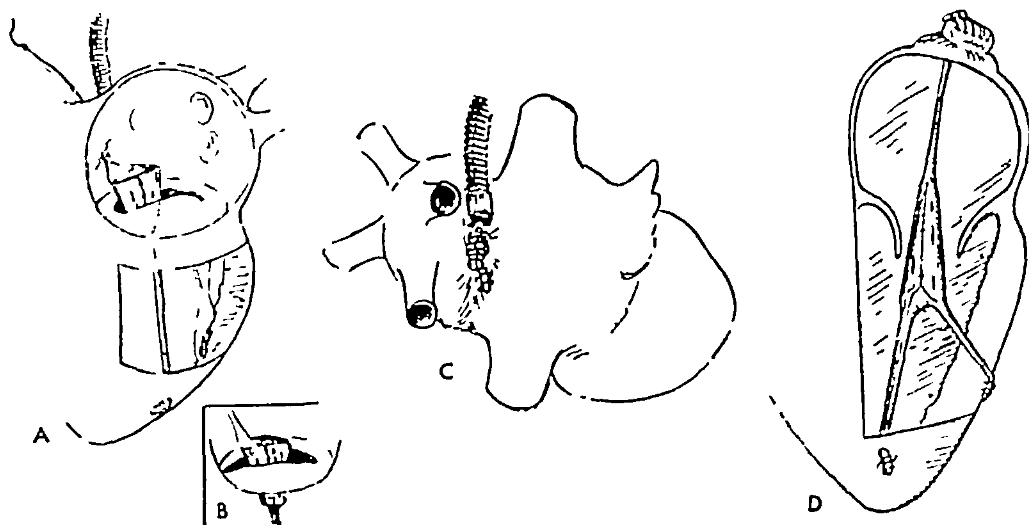


Fig 19. *A*, Further traction draws the stent gradually into the incompetent portion of the valve aperture When the palpable regurgitant jet has been greatly reduced or even abolished, the apical tail which now appears emerging from the ventricular puncture site is fixed to the ventricular epicardium by penetrating it with the needle-bearing end of the tied purse string *B*, In the arcuate form of mitral insufficiency, the cross section of the stent is made to resemble that of a geometric "segment of a circle on a chord" in order better to tamponade the leak *C*, The basal "tail" of the stent is anchored to the right side of the atrium by piercing it with the sutures used to approximate the edges of the separated interatrial groove. *D*, Alternate method of placing stent so that the larger end lies within the ventricular chamber.

given to intrapleural hemostasis bilaterally Both pleurae are drained by multi-windowed intercostal catheters inserted through posterior axillary stab wounds and connected with water-seal drainage systems The ribs are approximated with pericostal chromicized catgut sutures Continuous chromicized catgut is used to approximate the muscles and fasciae, and continuous wire is employed for the skin.

APPLICATION OF A PERICARDIO-CARTILAGINOUS STENT IN AORTIC REGURGITATION Transvalvular stents are recommended in aortic regurgitation only when there is an appreciable element of coexisting stenosis, and never when the incompetence is due primarily to annular dilatation. Since the stent is to be used only for aortic insufficiency accompanied by stenosis, it is essential that the stenosis be relieved first in order that the valve opening be rendered large enough readily to tolerate the implied surgical reduction in

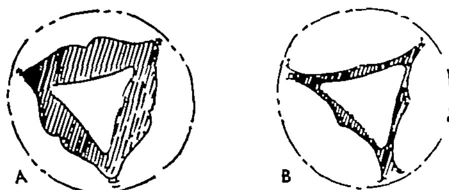


Fig 20 *A*, Relationship between diminished triangular aortic orifice and the transvalvular triangular pyramidal stent used in aortic insufficiency combined with stenosis. Systole. If no valvular flexibility is present the stent technique is contra indicated. *B* During diastole the valve leaflets should fit snugly against the appropriate level of the cross section of the stent.

orificial size. This requires a preliminary digital exploration using the supravalvular approach through the aorta and a pericardial or other type of pouch. It is obvious that such a stent cannot be tolerated within the aortic orifice unless a significant amount of valve mobility is present or can be restored (Fig. 20*A*, *B*).

Both pleurae are opened, and the sternum is transected using a transversely scalloped double inframammary incision. Both sets of internal mammary vessels are divided and ligated. Most of the portion of the left side of the pericardium which lies anterior to the left phrenic nerve is resected for the preparation of the stent. A preserved homograft of pericardium is used to prepare the aortic pouch, and its central opening is attached by imbricating mattress sutures to the edges of the incision made in an excluded portion of the ascending aorta. By the use of a purse-string suture, it is converted into a water-tight pouch.

The operator's ungloved left index finger is inserted into the pouch, and as the aortic clamp is released it slips forward to enter the aortic lumen. It is advanced to the valve level to evaluate its condition and to maximally relieve the coexisting stenosis and establish an adequate sized valve opening. It is

essential that as great a degree of valvular mobility be established as may be possible Instrumental help may be required.

After the maximal opening of the valve has been achieved, the residual or resulting "aperture of incompetence" (remaining aperture during diastole) is evaluated with respect to size, shape and condition of the valve margins. This aorticoventricular fistula almost never is larger than that which can be blocked by inserting a finger tip. The desirability of the use of a stent, the appropriate effective size and shape for its cross section, and its relative degree of tapering,

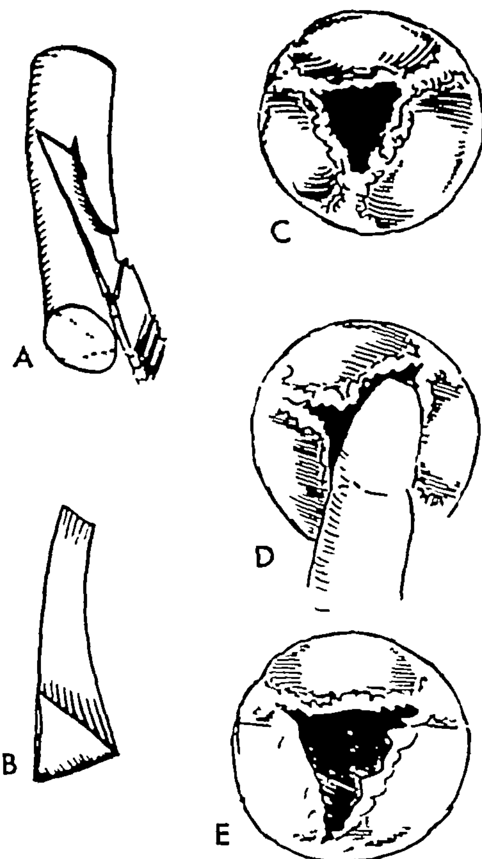


Fig 21 *A*, Carving or sculpturing segment of costal cartilage into tapering stent which usually is shaped like a triangular pyramid (trylon) *B*, Prepared trylon (truncated) *C*, Original stenotic aortic orifice, usually triangular in outline *D*, Digital or digitally guided instrumental commissurotomy is performed *E*, Finally established valve aperture is evaluated digitally for size, shape and mobility of the leaflets before preparing the cartilaginous stent

and appropriate length are determined. The finger is removed from the aorta which is clamped close to the base of the pouch. The latter is flushed with heparin solution as the finger is removed.

The major portion of the left fifth costal cartilage is excised subperichondrially. It is sculptured or carved in the same general manner as described for the preparation of the mitral stent Generally it is made somewhat longer (5 cm.) and it usually is made triangular on cross-sectional outline. Sometimes the valve opening requires that the stent provide an ovoid or irregular contour (Fig. 21*A*, *B*, *C*, *D*, *E*)

Again the stent is incorporated within a free pericardial tube with multiple perforations placed to prevent blood or serum collection and swelling about

the cartilaginous "core." Two pericardial tails are created at the wider basal extremity of the graft, while a single heavier one extends from its apex Heavy nylon sutures are attached to the free extremities of each of the three tails

A purse-string suture is placed about a relatively avascular area on the lateral surface of the left ventricle just above the apex. The operator's ungloved left index finger is reinserted into the aortic lumen by way of the pouch. The operator's right hand inserts the terminally threaded tip of a semimalleable probe through the circumscribed area of the ventricular wall into the lumen. The probe is advanced until the threaded end passes through the aortic valve orifice. The intra-arterial finger tip recognizes it and guides it until it emerges from the pouch along the palmar surface of the finger The suture, borne in the terminal eye, is grasped and held while the probe is removed.

The suture loop, which emerges from the pouch, is divided and one end is attached securely to the heavy apical extremity (or tail) of the graft. Traction upon the appropriate apically emerging suture draws the graft successively into the pouch, into the aorta and into the valve, the apical end of the cartilage coming to lie within the left ventricular chamber In order that the cartilaginous mass may enter the aortic pouch without causing severe blood loss, the air lock principle is employed, using an encircling strip of unrolled gauze for a secondary (more proximal) zone of hemostatic constriction about the pouch (Fig *22A*, *B*, *C*, *D*)

Finally, as the purse string is loosened somewhat, the cartilaginous portion of the stent enters the aorta and becomes inserted into the narrowed but surgically mobilized valve aperture. By alternate traction upon the basal and apical suspending sutures, the stent is "jockeyed' into the position of maximal filling of the "aperture of incompetence.' It is essential that valve flexibility be sufficient to permit this to be accomplished without the simultaneous production of a significant or serious degree of aortic stenosis. Sometimes a compromise must be accepted when the valve aperture cannot be greatly enlarged or when mobility of the elements is greatly restricted. When the optimal level has been established, the apical pericardial tail is transfixed by the needle-bearing end of the ventricular purse-string suture after preliminary hemostatic tying

The graft is reexamined by the intra aortic finger tip to determine its relations to the intra-aortic structures and a decision is made as to the best sites for the aortic suspension of the base of the stent. The aortic wall is locally circumscribed at selected points anteriorly to the left and posteriorly to the right with two purse-string sutures of No 3-0 cotton. Through each of these circumscribed areas large curved needles with heavy silk swaged on' sutures are passed successively into the aortic lumen to emerge from the pouch alongside the operator's finger

Each of these transaortic sutures is attached to the appropriate basal tail. Then, by traction upon the trailing ends of the sutures placed through the aortic wall, the pericardial tails individually are drawn into the aortic lumen and caused to penetrate its wall. After the operator becomes assured that

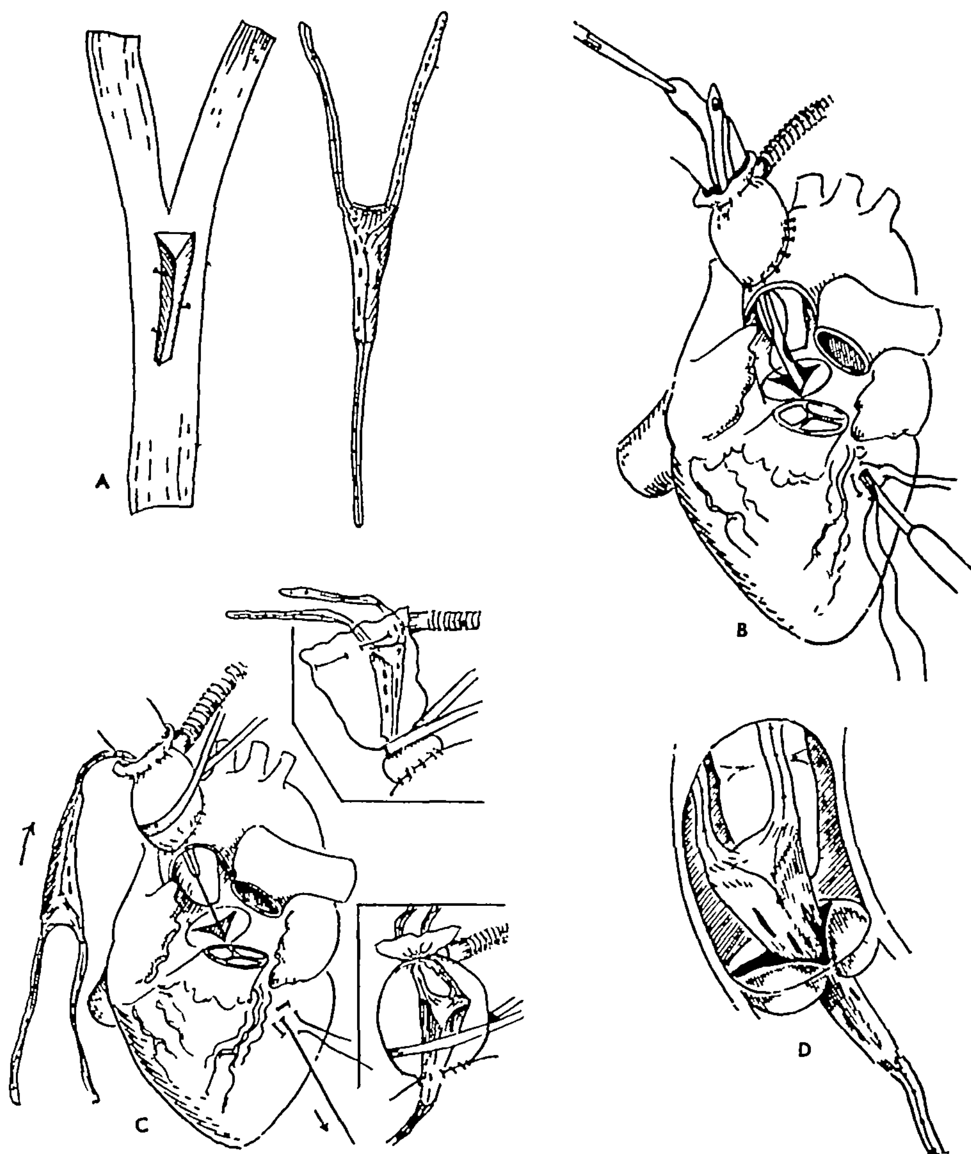


FIG 22 *A*, Preparation of the aortic pyramidal stent One heavy apical tail of pericardium and two smaller basal tails are necessary. *B*, Transventricular passage of terminally threaded probe through the left ventricular chamber and the incompetent aortic valve to emerge from the artificially created pouch on the palmar surface of the operator's index finger *C*, Apical end of stent being drawn into aortic lumen using "air-lock" technique on pouch (insets) to avoid causing serious blood loss *D*, Cartilaginous portion of stent fitting into valve orifice at that level of its cross section which accurately occludes the "aperture of incompetence."

valve function has been rendered and remains maximal, the pericardial tissue emerging from the aortic wall punctures is transfixed and attached to the respective purse-string sutures which first have been tied. The double basal suspension acts to prevent any rotation of the sculptured stent within the usually triangular valve orifice which might tend to destroy the "custom fit."

The aorta is reclamped, the pouch is excised and the aortic wall is repaired with one row of mattress and one row of simple continuous sutures of No. 4-0 arterial silk.

Both pleurae are drained by intercostal posteriorly placed multiperforated catheters. The sternal ends are drilled and brought together by braided

tantalum wire sutures (No 0) The ribs are approximated by heavy pericostal sutures of chromicized catgut. The muscles and fasciae are approximated by continuous catgut. The skin is closed with steel wire.

SURGICAL CONSTRICTION OF THE AORTIC ANNULUS FIBROSUS. The entire difficulty encountered in constricting the aortic ring in the treatment of insufficiency due to annular *dilatation is technical. This is related both to the* relatively deep location of the aortic valve within the substance of the heart and to the proximity and vital importance of the adjacent vascular structures (both coronary arteries, pulmonary artery, both atria) It is essential that the constricting sash should pass freely under both coronary arteries at a safe distance from their ostia. Also, the sash must course between the aorta and the overlying pulmonary conus. The ligament of the conus provides an excellent landmark for the determination of the proper line of dissection and offers a strong point of fixation which prevents the sash from tending to migrate upward and thus possibly to cause kinkage of the main coronary arteries Posteriorly, the sash must be placed beneath the level of the posterior atrio-aortic junction and, hence, usually must pass through both atrial chambers Necessarily then it must penetrate the intervening interatrial septum. However, it is sometimes possible to pass it between the aorta and the medial atrial walls without entering either atrial chamber While the method of constriction of the annulus has been reported previously,[8 21] it has since undergone certain technical modifications which have rendered it both safer and easier to establish.

When annular constriction has been contemplated in advance, the thoracic incision should be made across the entire anterior thoracic wall, dipping in scalloped fashion below both breasts and extending up into the apices of both axillas. The third right and the fourth left intercostal spaces are opened. The sternum is transected at the level of the fourth costosternal junction.

When a decision to apply aortic annular constriction is made only after the right thorax alone has been invaded, the incision simply is extended across the midline, the sternum being transected.

The mediastinal areolar tissue is wiped away from the underside of the sternal fragments, and the pericardium is incised transversely at the level of the sternal division. Care is taken to avoid injuring the phrenic nerves. A second incision, made in the central portion of the upper *pericardial segment* at a right angle to the former, aids in the exposure. The aorta and the outflow tract of the left ventricle are palpated carefully to recognize and evaluate the amount and location of any systolic thrill. At the same time, the surgeon estimates the vigor of the diastolic thrill, and attempts to palpate the shock of aortic leaflet closure, if any

It is preferred and recommended that a *pericardial or a plastic fabric* patch be prepared as described previously and be sutured to an incision made in an excluded portion of the right side of the ascending aorta. It is converted into a seamless pouch by tightening a purse-string suture of heavy nylon which is placed about its periphery in such a way that the line of the loops of the purse string courses at a greater distance *from the extremities* of the incision made in the patch than it does *from the sides* Care is taken that

only very short loops of suture material will come to lie within the lumen of the artificial pouch.

The operator, or an assistant, now may insert an exploring finger within the lumen of the aorta to examine the valve. Subsequently, this will be repeated to determine the amount of annular constriction to be established by the tightening of the sash to be placed about it.

The first definitive surgical step is the dissection of the lower portion of the pulmonary artery and the upper portion of the aorta. The preventricular branch of the right coronary artery will be encountered and usually must be divided between clamps and ligated at both ends.

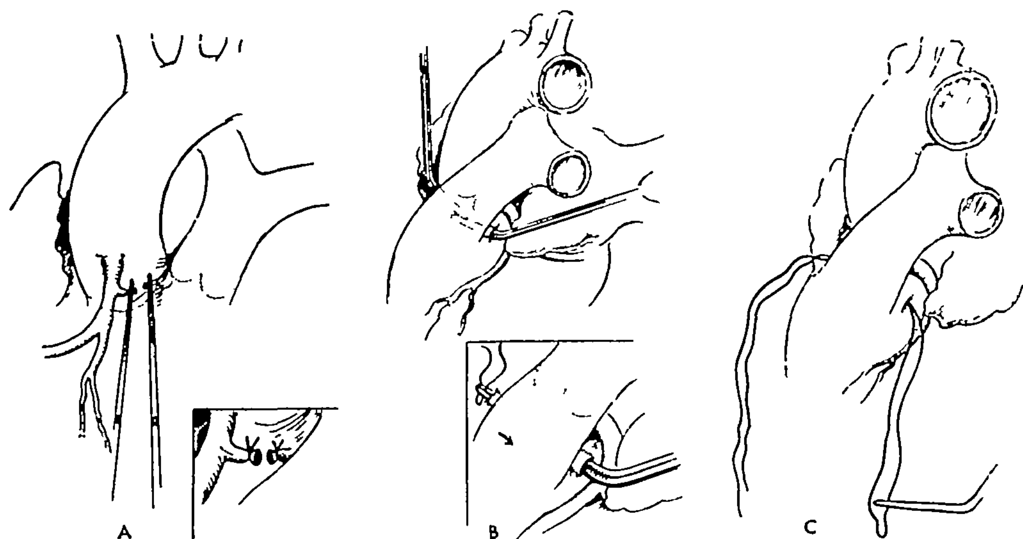


Fig 23 *A*, The preventricular branch of the right coronary artery (or the independent vessel when it arises from an independent aortic ostium) is divided between clamps and the ends are ligated The pulmonary artery and conus are dissected free from the aorta until the conus ligament is exposed *B*, Dissection is carried out beneath the conus ligament to completely separate the conus from the aorta Dissection from the opposite (posterior) side, beginning just anterior to the main left coronary artery, aids appreciably in mobilizing the conus *C*, Finally, a double suture of nylon is drawn through the path of dissection between the conus and the aorta

The ligament (or tendon) of the conus must be recognized but not injured, the definitive separation being carried out below it. The left main coronary artery is recognized running about a centimeter posterior to the left side of the pulmonary artery The epicardium is incised longitudinally for a distance of about 6 mm. along a line midway between the left coronary and the pulmonary artery Dissection is performed through this epicardial wound, the blades of the forceps being directed forward between the pulmonary artery and the aorta Soon it will be found possible to pass a clamp completely between the conus and the aorta at a level which lies just below the intact ligament of the conus. A loop of heavy white nylon suture is drawn through this tunnel of dissection and the appropriate ends are clamped loosely and held for subsequent utilization (Fig 23*A*, *B*, *C*).

Now a pair of angulated dissecting forceps or curved Metzenbaum scissors is inserted through the same epicardial incision and is directed posteriorly to pass under the left main coronary artery. The dissection is caused to advance

cautiously, in "mole-like" fashion, under the epicardium and close to the aorta, itself, always progressing in a posterior direction.

The right auricular appendage is clamped at its base and a purse-string suture of heavy (No 2 braided) silk swaged on a curved, round-edged needle is placed about its substance at a level which lies just distal to the clamp The tip of the appendage is incised for a distance of 1.5 cm. in a direction parallel to the blades of the clamp The excluded portion of the appendage is flushed with saline, and any obstructing trabeculae are divided. A Rumel tourniquet is applied to the extremities of the purse-string suture.

The interatrial groove is dissected for a distance of 1 5 cm., and the separation is deepened perhaps to an equal distance. A purse-string suture of heavy nylon attached to a swaged-on curved needle is placed about the area of dissection, making sure that each bite of the needle enters the lumen of the respective atrial chamber These sutures are placed so that they begin and end at the lower extremity of the line of dissection, hence, they are out of the way of the entering finger The ends of the suture are incorporated within a Rumel tourniquet. The tip of the operator's ungloved left index finger is inserted, palmar surface upward, into the dissected groove and a large bladed knife is laid upon it. The left half of the partially split interatrial septum is incised and the operator's finger tip seeks out this opening and passes through it entering the left atrial chamber (see Fig 16*B*, *C*)

The operator's right hand now guides a terminally threaded (with heavy white nylon suture) semimalleable curved probe under the mobilized left coronary artery and along the dissected tissue tunnel, keeping close to the aorta. Finally, the probe tip reaches the left atrial wall at the posterior aspect of the aortic circumference. The intra-atrial finger tip determines the highest feasible site of atrial wall entrance and aids in the placement of the puncture of the probe tip into the atrial chamber Sometimes instead of actually breaking into the atrial lumen, a properly curved probe will simply follow the outer surface of the aorta, passing between it and the covering lining layer of atrial musculature. This deviation is preferred over what may be considered as the orthodox technique. In the latter, once the probe tip has entered the left atrial chamber, the handle is turned over to an assistant and the operator's right index finger, in turn, is divested of its glove covering.

The ungloved right index finger tip enters the incised tip of the right atrial appendage and passes to the region of the interatrial septum which lies just above the beginning of the aorta. It makes contact with the apposed intracardiac tip of the left index finger on the opposite side of the septum. The assistant advances the probe tip cautiously, the operator's intracardiac index finger guiding it (and if necessary bending it somewhat to penetrate the interatrial septum as near as possible to the aortic wall) The tip of the probe continues to advance until it emerges from the right atrial appendage along the right index finger The suture borne in its terminal eye is picked up by an assistant and is held. Then the probe is withdrawn from the heart leaving a loop of nylon suture traversing both atrial chambers and piercing the intervening septum (Fig 24*A*, *B*, *C*)

With the interatrial fingers still in place, an experienced assistant now dis-

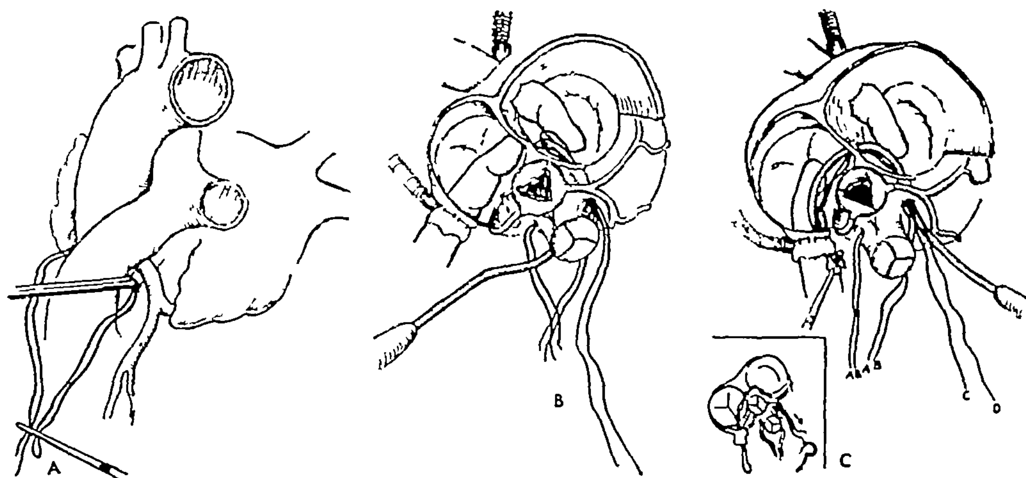


Fig 24. *A*, Dissection with a blunt hemostat beneath the left coronary artery is directed backward close to the wall of the aorta and toward the left atrial chamber. *B*, The operator's left index finger is inserted into the left atrial chamber by way of the dissected interatrial groove A semimalleable terminally threaded probe is passed under the left coronary artery following the dissecting tunnel until it breaks into the left atrial chamber in close relationship to the aortic wall Sometimes it may follow the aortic wall closely, passing between it and the medial atrial wall The operator's right index finger now is inserted into the right atrial chamber by way of its appendage. By approximating the two finger tips against the atrial septum, the desired site of septal penetration is determined *C*, The probe is advanced by an assistant, following the guidance of the intracardiac fingers, and its tip pierces the interatrial septum It may be bent within the heart to facilitate its further passage and finally its emergence from the right auricular appendage. The suture borne in the terminal eye is grasped and held while the probe is withdrawn from the heart (inset).

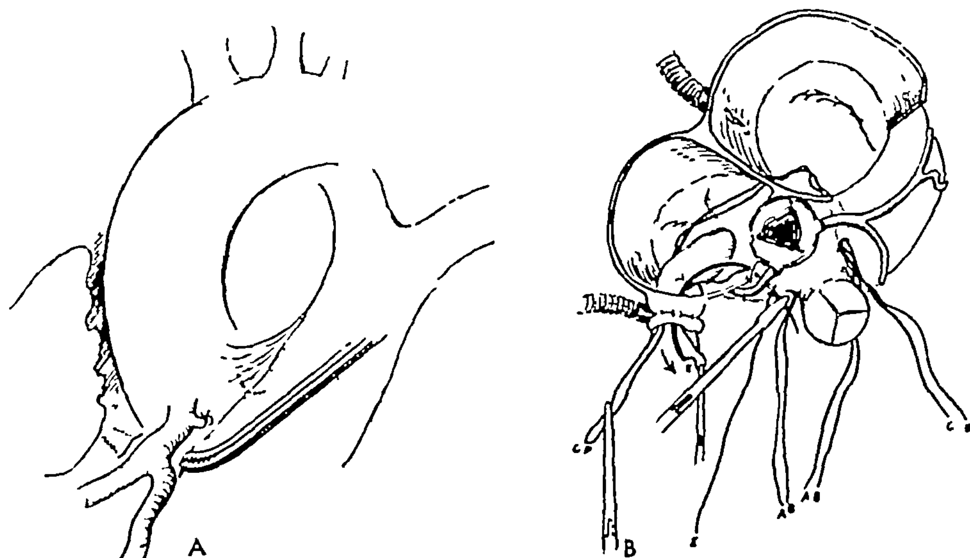


Fig 25 *A*, Dissection is carried out under the right coronary artery and is directed toward the right atrial chamber *B*, The blunt end of a large curved Atraumatic needle is passed through the path of dissection under this vessel and penetrates the right atrial chamber from which it and its attached black nylon suture emerge along the palmar surface of the inserted finger Together the three passed sutures now surround the aorta at the annular level.

sects the tissues under the right coronary artery A curved, round-edged suturing needle swaged on a heavy black nylon suture is passed, usually in reversed fashion, under the right coronary artery to enter the right atrial lumen close to its junction with the aorta. The needle is caused to emerge from the appendageal opening along the palmar surface of the intracardiac finger The suture is drawn through and the needle is cut away (Fig 25*A*, *B*)

A heavy sash of nylon fabric is prepared by cutting No 14 nylon material in a strip 4 inches (10 cm.) in width and about 23 inches (60 cm.) in length. Recently we have been using parachute nylon* (Navy specification #MIL-C-7020-B type) for this purpose because of its greater integral strength. The ends are cut to taper, and the extremities are attached securely (by suturing) respectively to the right atrial extremity of the black nylon suture (the one which passes under the right coronary artery) and to the right atrial extremity of one of the nylon strands obtained by dividing the white loop which has traversed both atria and the intervening septum. The other strand is preserved temporarily as a spare' to be used if the sash should become detached from the first one. Smooth but intermittent traction is made upon the appropriate nylon extremity protruding from under the left coronary artery, and the attached end of the sash is gradually drawn into the right atrium along the right index finger It pierces the interatrial septum and, perhaps aided by manipulations of the intracardiac left index finger, traverses the left atrial chamber, finally exiting from it to enter the "tunnel of dissection" and to appear at the site of epicardial incision just anterior to the left coronary artery It is essential that about one half of its actual length should emerge from under the left coronary artery The "spare transatrial nylon strand is now removed from the heart. The nylon strand attached to the leading end of the sash is tied (by use of a half-hitch knot) to one of two obtained by dividing the suture loop which has previously been passed under the pulmonary conus Its remaining fellow strand is preserved for possible later use.

The trailing end of the nylon sash is now caused to enter the right atrial chamber (along the finger) and to emerge from the tunnel of dissection under the right coronary artery by traction upon the trailing end of the black suture appearing just to the left side of this artery Similarly, traction upon the right extremity of the appropriate strand of white nylon which passes under the pulmonary conus causes the lead end of the nylon sash to follow its course under the pulmonary conus and to emerge just to its right side in close proximity to the trailing end of the sash (Fig 26*A*, *B*, *C*)

If it is deemed sufficient to constrict the aortic annulus merely by tightening and tying this sash with the appropriate amount of tension, this is carried out immediately Dependence is placed upon a finger inserted, via the pouch, into the aortic lumen to determine the exact amount of tightening required to restore full competence and to overcorrect the lesion without producing a serious degree of narrowing of the aortic outlet passageway It is urged that the special double bowknot, illustrated in Fig. 29*B*, *C*, *D*, be employed in order that all constriction may be released instantaneously should a sudden weakening of cardiac contractions necessitate it.

* Obtainable from New Market Manufacturing Company, New York, New York.

However, the authors have begun to suspect that sometimes the posterior (intra-atrial) portion of the sash may be placed over only the most extreme lower edge of a highly placed (relative to the atrial chambers) aortic annulus (Fig. 27*A*). In such an eventuality, it is conceivable that it might tend to migrate downward (toward the apex) and to "slip off" the annulus, coming to lie over the left ventricular outflow tract but no longer providing corrective compression to the region of the annulus (Fig. 27*B*). Such an eventuality might explain the experience of one of our early patients who exhibited an excellent initial restoration of valve function (elevation of the diastolic blood

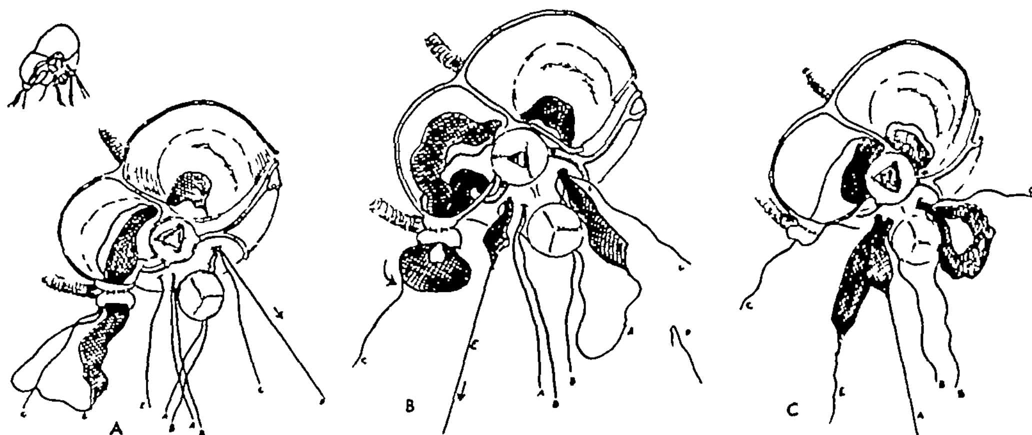


Fig 26 *A*, A 24 inch (60 cm) long, 4 inch (10 cm) wide strip of parachute nylon is trimmed to taper to a point at either extremity One end is attached to the right extremity of one of the strands obtained by dividing the long transatrial suture loop By traction upon the opposite end (taking care not to "saw" into the left main coronary artery), the "sash" of nylon is caused to enter the heart, to traverse both atrial chambers and the intervening atrial septum, and to emerge just anterior to the left coronary artery *B*, After one half of the sash has emerged from the heart, the trailing end is attached to the black nylon suture emerging from the right atrial appendage and is drawn into the atrial chamber, through its wall It finally emerges to the left of the right main coronary artery *C*, The lead end of the sash is attached to one strand of the divided white nylon loop encircling the pulmonary conus, and is drawn under it to emerge anteriorly between the conus and the anterior aspect of the aorta In order to facilitate subsequent steps, it is better at this time to leave a free loop of sash protruding to the left of the conus as shown The long unused white nylon suture strand extending through both atria is now removed The shorter extra one under the pulmonary conus is preserved

pressure, disappearance of the diastolic murmur), only to lose it within a few days.

For this reason, the authors now feel that in patients with a low attachment of the atria to the aorta posteriorly, a second constrictive sash should be placed under both coronary arteries and the conus ligament, but that posteriorly it should lie external to the atrial lumina. While such a sash would tend, upon the application of constriction, to "rise" upon the posterior aspect of the aorta, it readily may be bound inseparably to the internal sash by including it within an encircling heavy nylon suture (or two) which first has been passed around the contiguous intra-atrial portion of the internal one It readily can be seen how a much broader zone of constrictive pressure may be brought to bear posteriorly upon the underlying annulus by the use of such

a double sash technique (Fig 27C) Such a double sash would be especially appropriate in a luetic case with a degree of associated aortitis and dilatation of the first portion of the aorta.

Application of the Second (External) Constricting Sash. When a second constricting sash is to be placed, it is very desirable that the loop in the lead end of the first sash which is shown to the left of the pulmonary conus in Fig. 26C should not be drawn through completely until after the passage of another nylon traction suture under the left main coronary artery If the sash had been drawn flush previously, a great deal of difficulty might be experienced in passing this new suture accurately through the same "tunnel of dissection."



Fig. 27 *A*, Sectional drawing indicating possible precarious placement of posterior sash in relationship to a high lying annulus fibrosus *B*, It would seem possible that the posterior intra-atrial portion of the constricting sash might migrate downward so that constriction would become applied only about the left ventricular outflow tract with consequent loss of valvular competence. *C*, Sectional drawing indicating greater surface of compression applied to annulus when both an internal and an external sash are used posteriorly

The first step in the placement of a second sash is the encirclement of the intra atrial portion of the internal sash with a suture (or two) A small, curved, round-edged needle swedged on a heavy black nylon suture (No D54-39*) has proven most satisfactory for this purpose. It is passed, somewhat diagonally to the course of the internal sash, through the upper portion of the left atrial wall just to the right of the posterior wall of the aorta and to the left of the interatrial septum, in close proximity to the atrio-aortic junction. The intracardiac (left) index finger guides the passage of the needle tip around the rolled up sash so that its substance does not become penetrated. The needle point then repenetrates the atrial wall from within, and the inclusion of the sash within the suture loop is completed (Fig 28A) The ends of the suture are kept long for future reference.

A threaded probe (or a suturing needle) is now passed under the right coronary artery emerging just posterior to it, and the suture is held as the probe is withdrawn (Fig. 28B) A similar probe is passed under the left coronary artery, emerging well posterior to it. The suture it bears is similarly held as the probe is withdrawn (Fig 28C)

* Obtainable from Ethicon, Inc. New Brunswick, New Jersey

The tapering tip of a second nylon sash, made similar to the first one, attached to the posterior extremity of this suture loop, and about half of length is drawn under the left coronary artery, emerging anteriorly. T suture in the lead end of the sash is now tied to the remaining white sutu

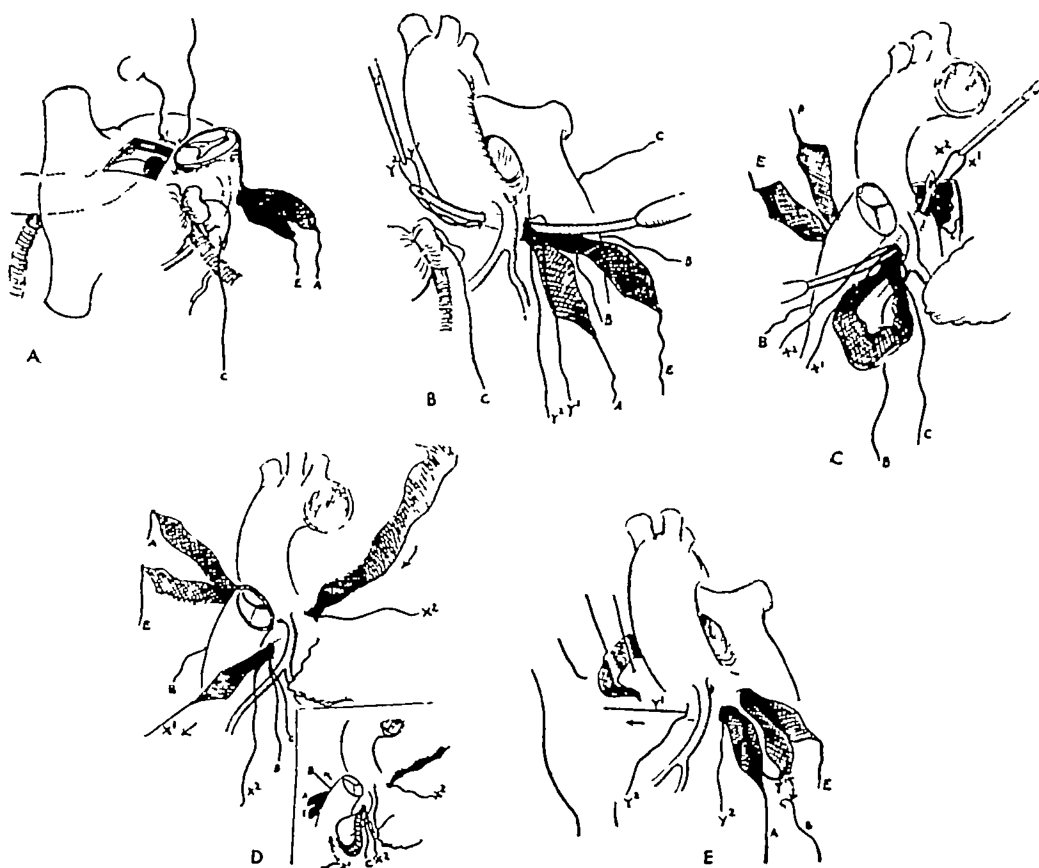


FIG 28. *A*, With internal guidance of the left index finger placed within the left atrial chamber, a small, curved, suture-bearing needle may be passed through the upper atrial wall to the right of the aorta to encircle the intra-atrial portion of the sash *B*, A threaded probe is passed under the right coronary artery but is directed superficially so that it does not enter the right atrial chamber but emerges externally between the right atrium and the aorta The suture loop is grasped and the probe is withdrawn *C*, Similarly, facilitated by the presence of the protruding left loop of sash, a threaded probe is passed under the left coronary artery and is caused to emerge externally between the left atrium and the aorta This suture also is grasped and held while the probe is withdrawn *D*, One posterior extremity or strand of this divided suture loop passing under the left coronary artery is attached to the tapering end of another nylon fabric sash, and it is drawn under the left coronary artery for one half of its length *E*, The anterior extremity of the second sash is drawn successively under the conus and then under the right coronary artery by repeated attachment to the appropriate leading sutures.

which passes under the pulmonary conus, and which has been preserved for just this purpose (Fig. 28*D*). By traction upon the right extremity of this suture, the sash is drawn between the pulmonary conus and the aorta, emerging to the right of the former structure Its lead suture is now tied to one of the strands of the loop recently placed under the right coronary artery so that the end of the sash may be drawn to emerge to the right of this vessel (Fig 28*E*).

The trailing end of the second sash is now brought around posterior to the aorta and is laid between the strands of the nylon suture which has been placed to encircle the internal sash (Fig 29*A*) Tying of an ordinary bowknot in the nylon suture will maintain near approximation of the internal and external sashes both during their actual tying down and subsequently

Both intracardiac fingers are removed and the atrial openings are repaired respectively by tying down the purse-string sutures and by oversewing of the lips of the respective wounds If an aortic pouch has been prepared and placed

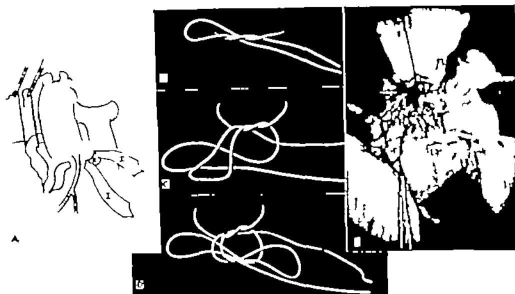


Fig. 29 *A* Finally both ends of the second (external) sash are brought into contact to the right of the right main coronary artery The ends of the first (internal) sash emerge together just to the left of this vessel. The suture encircling the intra atrial portion of the intracardiac sash is caused loosely to encircle the overlying portion of the extracardiac sash. *B*, Each of the sashes is tied in the special bowknot indicated above. While this is a secure square knot it may be readily untied completely by traction upon the appropriate free ends of the fabric. Usually, the deeper (intracardiac) sash is tied first. It is essential that overcorrection be accomplished and that a slight to moderate degree of aortic stenosis be established at this time. *C*, Beginning second throw in bowknot. *D* Completion of bowknot. *E*, Photograph of finally tightened aortic annular constriction. Ends and loops of special bows must not be trimmed. Then should later surgery be deemed necessary either to loosen or further to tighten a bow it may be done readily simply by reopening the thorax.

previously, the left index finger of operator or assistant is inserted into the aortic lumen to guide and to limit the degree of constriction produced. Usually, the internal sash is tied down first, using the special double bowknot illustrated in Fig 29*B*, *C*, *D* It is tightened until the incompetence beco es overcorrected, a distinct aortic systolic thrill finally being produced. The intra-aortic finger will be able to limit the narrowing of the aortic passageway (in the average male adult this should not be made less than a 1½ finger orifice). It is wise to hold the first throw of the special bowknot placed in the sash at the selected degree of constriction for a few moments before placing the second throw Should the heart action at any time become weak, either the single or double throw bowknot may be released instantaneously by traction upon the free ends.

After tying the internal sash, the external one is tied down in similar fashion. Sometimes it may be necessary to readjust the nylon suture binding it to the internal sash, but this is a very minor problem. The bowknot in one sash (external) lies to the right of the right coronary, between the aorta and the right auricular appendage. The other one (internal) lies to the left of the right coronary artery. The ends of both tied sashes are left in place without trimming. Providing they have been tied in true square knots, no loosening need be expected. If only a "granny" knot has been used, transfixion sutures must be placed through their substance to insure holding.

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DISCUSSION

Jesse Edwards (*Rochester, Minnesota*)

With respect to rheumatic mitral stenosis and mitral insufficiency, it is well to recognize that if we have some reduction in the size of the orifice of the mitral valve, from a pathologic point of view we say there is a degree of mitral stenosis If there is some regurgitation of left ventricular blood into the left atrium during ventricular systole, then we are forced to say that there is some mitral insufficiency

That is looking at things perhaps in too rigid a manner The practical point is that ordinarily a patient with mitral rheumatic disease presents a problem *either of mitral stenosis or of insufficiency rather than of difficulties from the* two conditions combined. We can therefore separate the two conditions as two distinct entities

I think there are two keys to the problem when mitral insufficiency exists, as follows. First, either the hinge of the door is frozen or, second, the door just isn t big enough to close the opening The first situation, the" frozen hinge, is usually dependent upon calcification of the mitral valve at one or both commissures The commissures can be considered the hinges of the door, which is the mitral valve. Insufficiency results if the door is held in an *open state by commissural calcification.*

Now we come to a more common type of mitral insufficiency, the one which results from inadequate length of the leaflet. Usually it is a problem of the posterior leaflet being shortened and lying quite some distance from the anterior leaflet during ventricular systole. When there is dilatation of the left atrium, the annulus fibrosus of the mitral valve is dislocated away from the mitral orifice. When this happens, the posterior mitral leaflet is pulled away from the orifice, thus accentuating the insufficiency caused by shortening of the mitral valve resulting from intrinsic rheumatic disease. In this type of case we have a combination of fibrosis of the valve in which the valve leaflet becomes shortened, setting up a stage for regurgitation, then regurgitation itself, with dilatation of the left atrium, causes the posterior leaflet to be pulled away more from the orifice, and a vicious circle is set up One way of overcoming mitral insufficiency of this type is to overcome the dilatation of the lowermost portion of the left atrium.

Alfonso R. Albanese (*Buenos Aires*)

When the finger is in the atrium to explore the mitral valve, one can feel different kinds of jets associated with mitral insufficiency. However, in 60 per cent of the cases, it is located at the posterior commissure (Fig. 1*A*). The regurgitant jet can be eliminated by placing the finger in the commissure (Fig. 1*B*) or depressing the atrial wall over the commissure by pressure from

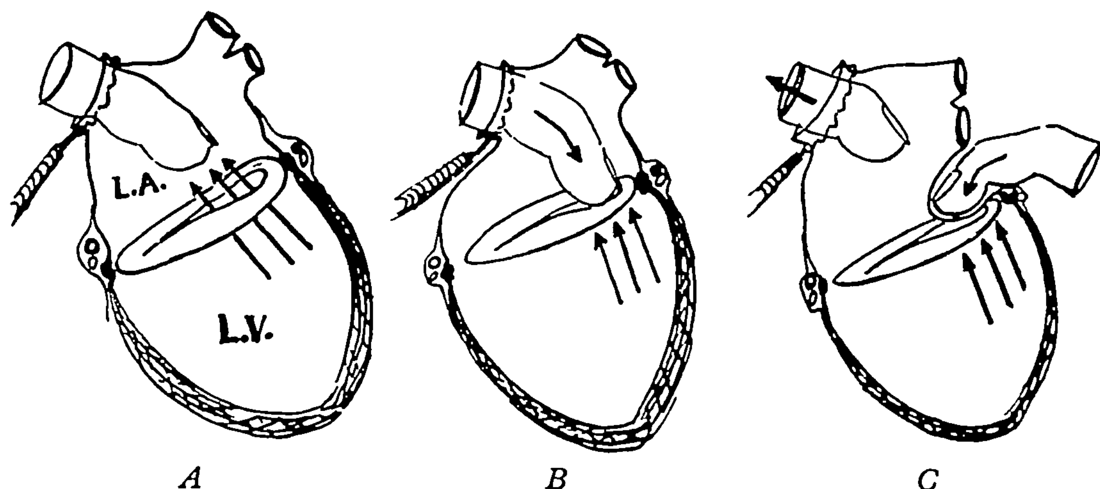


Fig 1 Diagram of mitral regurgitation and two ways of stopping the regurgitant jet

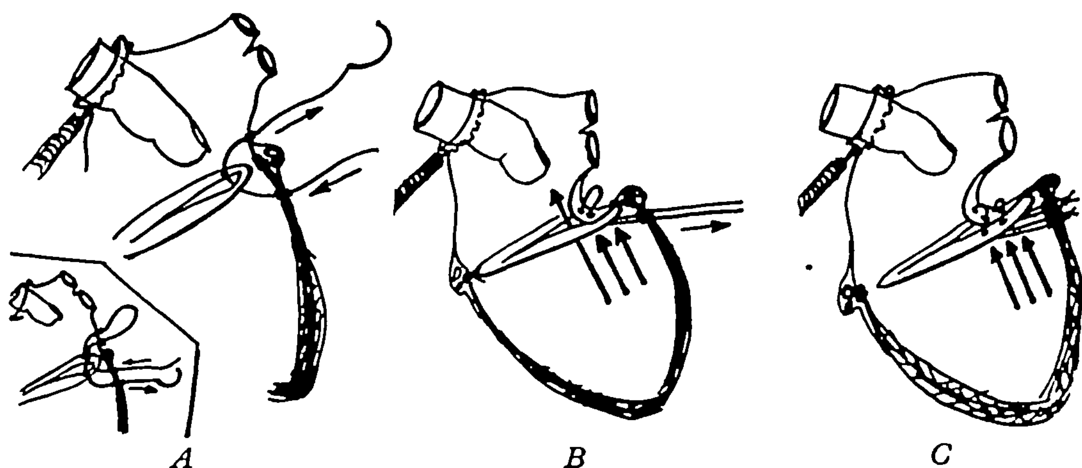


Fig 2 Steps in the operation of "posterior atriocommissuropexy"

without (Fig. 1*C*). I wondered if the latter maneuver could indicate a method for repair, and carried out a number of experiments on the dog.

A curved needle threaded with silk or nylon enters the wall of the left ventricle about 1 cm below the coronary vessels and behind the posteromedial commissure. The needle traverses the anterior valve leaflet and emerges from the atrium between the left inferior pulmonary vein and the atrioventricular sulcus (Fig 2*A*) The needle is then passed back into the atrium, through an edge of the posterior valve leaflet, and then out through the ventricular wall about 1 cm. from where it entered (Fig 2*B*). Additional sutures may be placed if traction on the suture does not abolish the jet (Fig. 2*C*).

I have carried out this operation of "posterior atriocommissuropexy" in one patient and the regurgitation was satisfactorily corrected.

J C. Davila (*Philadelphia*)

The concept of circumferential constriction or purse-stringing of cardiac valves for the relief of insufficiencies has been studied experimentally in the Cardiovascular Research facilities of Episcopal and Presbyterian Hospitals, which are under the direction of Robert P Glover, since 1951 The rationale

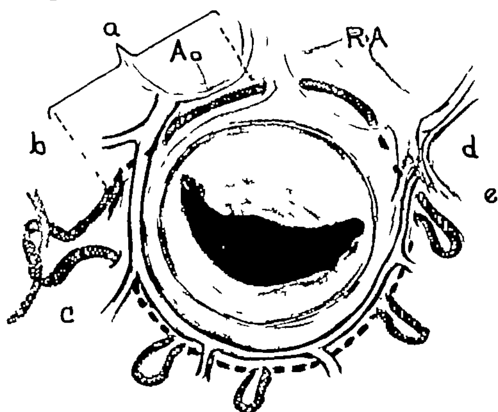


Fig. 1 Diagram of the mitral valve ring showing the relations of the circumferential suture. *a*, Segment of suture lying in the transverse sinus and beneath the circumflex coronary artery This segment of suture is covered with pericardium to prevent the suture from eroding through the base of the atrial appendage. *b*, Anterior descending coronary *c* Circumflex coronary *d* Coronary sinus. *e*, Posterior descending coronary

of its application and its anatomic basis first appeared in the literature in Surgery, Gynecology and Obstetrics in April, 1954 (Fig. 1) This principle applies to the diseased mitral valve because here the valve anatomically consists of an atrioventricular communication bounded by two rings The outer ring, the annulus, can be reduced in size to relieve cusp tension and to allow motion and coaptation of the leaflets without encroaching upon the effective opening of the inner ring, the mitral orifice.

At Episcopal and Presbyterian Hospitals during the past three years the following thorough studies have been carried out: (1) Dissections and trials of annular purse strings on both normal and diseased human hearts obtained at autopsy; (2) experiments in which 150 dogs have been used to date; (3)

experimental trial of various technical modifications culminating in the development of a simple and satisfactory technique; (4) electrocardiographic, hemodynamic and pathologic studies on experimental animals, and (5) recent studies and clinical application in 5 patients.

It has been demonstrated that this technique is feasible and safe. *When properly performed:* (1) It has been well tolerated by the dogs and the diseased human heart, (2) it can be done in the largest hearts, (3) with proper anatomic orientation it is not a difficult procedure, (4) experimentally and clinically the observable effects of mitral insufficiency can be reversed, (5) no significant damage to coronary or conduction systems or to myocardium is demonstrable,

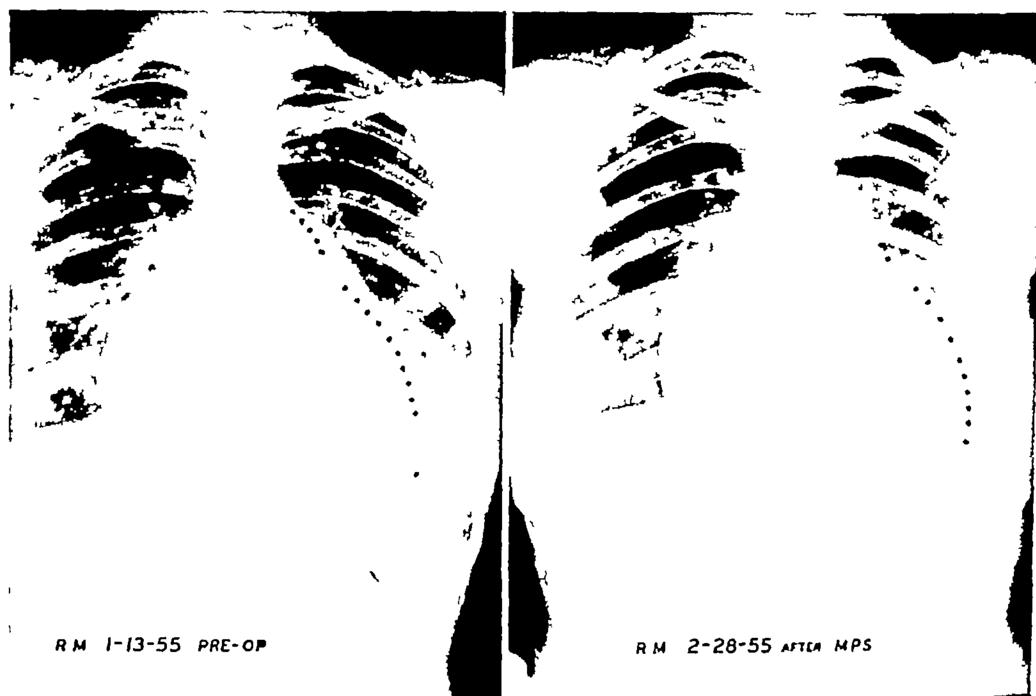


Fig 2 PA roentgenograms of the first case The left contour of the postoperative cardiac silhouette has been outlined with dots and this dotted contour has been superimposed on the preoperative film to demonstrate the degree of reduction in cardiac size noted approximately one month postoperatively

(6) dynamic changes which might indicate stenosis are not produced, (7) the suture does not cut through into the cardiac lumen as the operation is now performed although in our early experiments this was the most serious problem; (8) clinically the desired effect appears obtainable at least in those cases in which remaining valvular substance is reasonably pliable. This would appear to apply to the majority of cases. An additional point of interest in mitral purse string is the fact that such a procedure not only reduces or eliminates regurgitation but can prevent the progression of relative insufficiency by limiting the size to which the annulus can enlarge and thus avoid progressive increase in the magnitude of the regurgitant volume

In cases in which damage to valve cusps is of such degree that nothing short of replacement of valve elements can be expected to be of value, one may have to resort to prosthetics such as described by Harken, and recently modified

by Bailey Unfortunately the various techniques employed by these authors have shown generally unsatisfactory results

Within the last three months we have operated upon 5 patients The first 2 of these were operated upon over two months ago They have returned home and are markedly improved to date. The third patient has shown great improvement while still in the hospital. The fourth case is still too recent to evaluate. The fifth patient died at operation when irreversible ventricular fibrillation developed as the pericardium was opened. Two of these patients

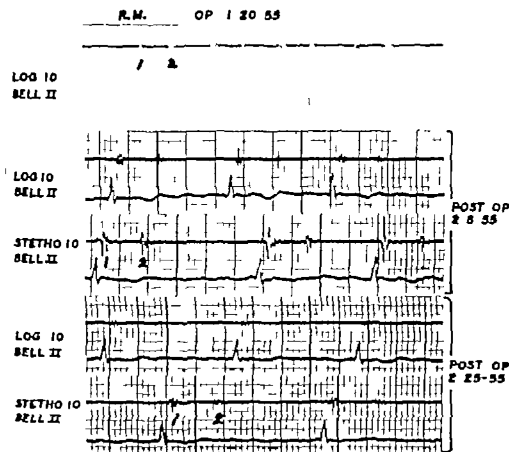


Fig. 3 Pre- and postoperative phonocardiograms of the first case. The preoperative tracing was taken by the referring physician. Note the almost total disappearance of the systolic murmur

had mitral commissurotomies at the same intervention just prior to placement of the encircling suture. Mitral insufficiency was the predominant lesion in these. Each of these 5 patients was in intractable congestive failure prior to surgery The illustrations show the pre- and postoperative roentgenograms (Figs 2 and 5), phonocardiograms (Figs 3 and 6) and left atrial pressure tracings (Figs. 4 and 7) in the first two patients. In one of these the heart size was reduced significantly within the first three postoperative weeks The phonocardiograms showed an almost complete disappearance of the murmur in both cases. The left atrial tracings showed a decrease in pressure from 78/15 mm. Hg with a mean of 35 to 17/7 mm. Hg with a mean of 11 in one

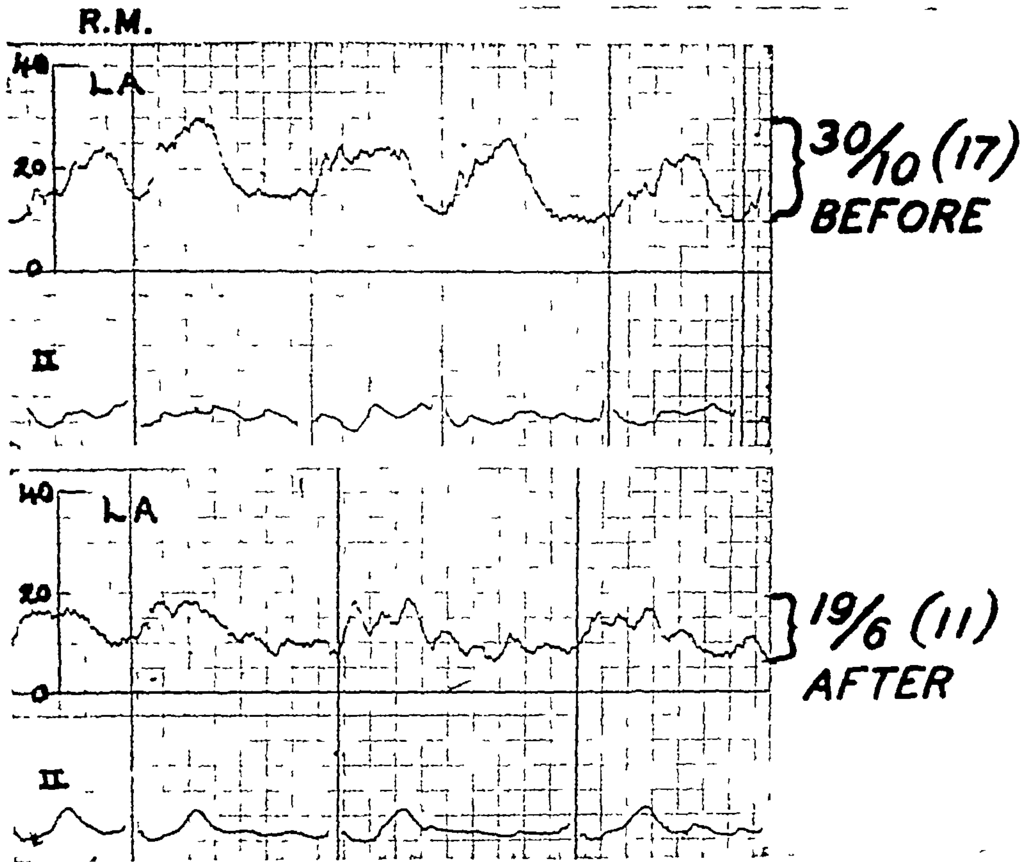


Fig 4. Tracings of the left atrial pressure pulse taken at operation before and after placement of the circumferential suture in the first case Note the drop in pressure mentioned

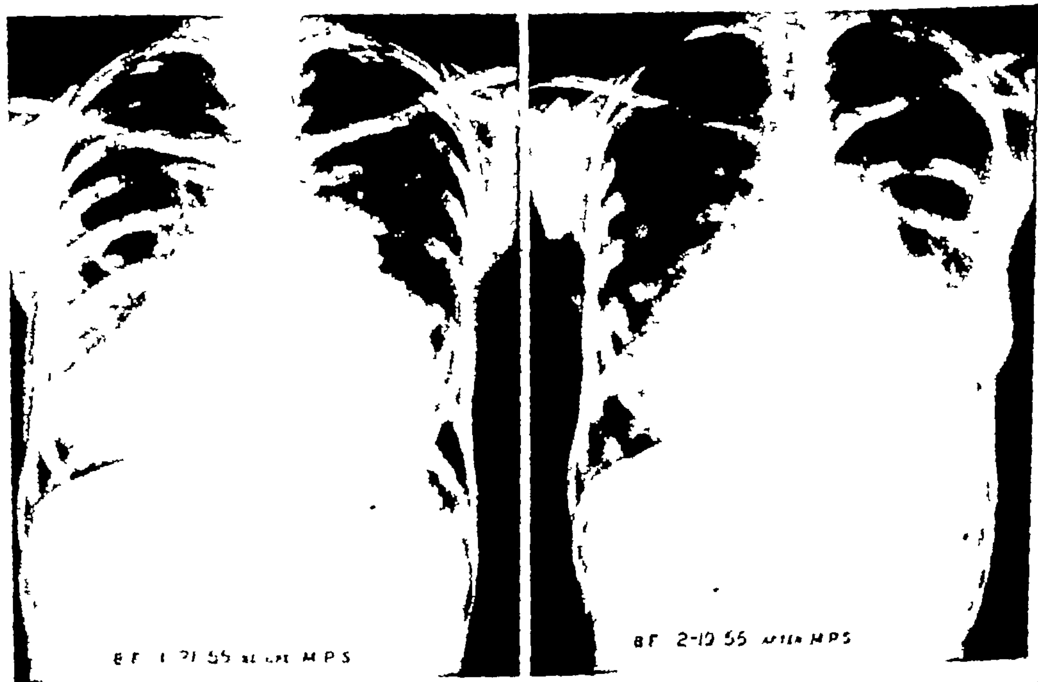


Fig 5 PA roentgenograms of the second case taken pre- and postoperatively Although there is a moderate amount of residual pleural reaction postoperatively, one can see a very slight degree of reduction of cardiac size by superimposing the actual films

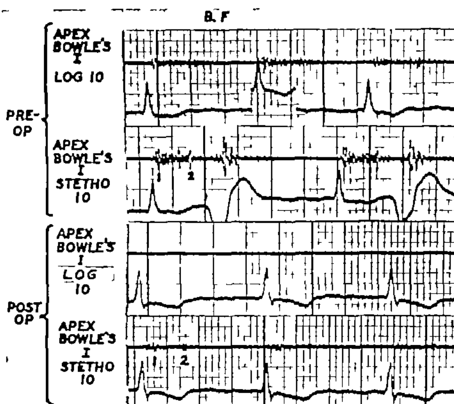


Fig. 6 Phonocardiograms taken pre and postoperatively in the second case show a clear diminution of the murmur as well as a decrease in the intensity of the second sound which was quite marked in the preoperative tracing

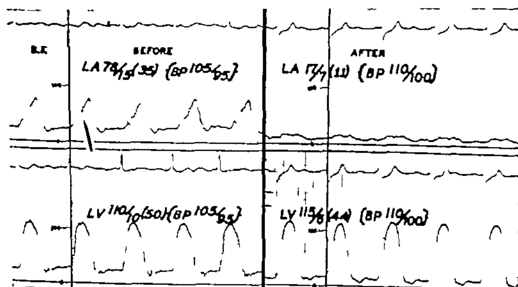


Fig. 7 Tracings of the left atrial and left ventricular pressure pulses taken at operation before and after placement of the circumferential suture. Note the remarkable drop in left atrial pressure as well as the changes in pressure and contour of the left ventricular tracing. The steeply sloping reduced ejection phase of the ventricular curve, seen before the procedure, changes to a more normal appearing ventricular peak. The ventricular diastolic pressure drops closer to a normal level.

case, and from 30/10 mm. Hg with a mean of 17 to 19/6 mm Hg with a mean of 11 in the second.

The purse-string principle has possible application in other valvular insufficiencies and also in septal defects. Sondergard, Bjork and Crafoord have shown its value for the closure of atrial septal defects. Many additional phases merit study, and we have been engaged in such animal experimental investigations for many months. The mitral and tricuspid valves, being similar anatomically, should lend themselves most readily to this type of procedure. In aortic valvular insufficiency, of nonluetic etiology at any rate, the degree of damage to cusps and the nature of the pathologic anatomy make it appear doubtful that significant improvement of the incompetence could be obtained by this method without producing serious reduction in the size of the effective aortic orifice. In this connection it is unfortunate that the pressure gradient across the aortic valve after encirclement in Dr. Bailey's two patients was not demonstrated; some information on this point would be of value at this time.

Ross Robertson (*Vancouver, B.C*)

The procedure that I suggest can be used only in a limited number of cases. It is useful when the mitral regurgitation and mitral stenosis are about equally important and when the valve is nearly normal except that the cusps are stuck together for about half the distance, and the posterior cusp is quite narrow. The anterior cusp is nearly normal.

In two such patients we have found, after dividing the commissure completely, that there was a severe jet of regurgitation. This could be stopped very readily by pressing the finger against the annulus opposite the posterior commissure.

As a result of this, we decided to try making a pedicle flap of pericardium and rolling up the end of the flap into a ball about the size of one's thumb. This is sutured over the outer surface of the heart just over the annulus in the region where one finds that pressure will stop the regurgitation.

Two patients have been operated on by this method with gratifying results.

CONCLUDING REMARKS

Dwight E. Harken (*Boston*)

Dr Davila's and Dr. Glover's operation is very interesting and challenging.

Dr. Albanese's operation, as you know, is quite similar to that by Dr. John Hayward, from Melbourne, which he mentioned something like two years ago, and his follow-up indicated that in all probability the sutures had cut out and the atrium had reverted to its normal position.

Dr. Bailey's presentation was fascinating to me, with its discussion of mitral regurgitation and other cardiac problems. I think there is surprisingly little difference between the Philadelphia "pyramid" and the Boston "baffle," and I think we can be somewhat skeptical about either one of them. Some of my baffles I did cover with everted vascular graft, which makes them even more like each other, and I do not believe my baffle procedures have stood the test of time. I think there is similarity, therefore, between the

covered cartilage and the covered Lucite baffle of various sizes and shapes that we have used.

There is very little difficulty technically in placing this posteromedially, though it may be simpler, as Dr Bailey has mentioned, from the right side. The technical difficulty was not, per se, the problem. The principles outlined have had their own disappointing aspects that require no further discussion

Charles P Bailey (*Philadelphia*)

I suppose it is just following pattern that Dr Harken and I should usually start from different viewpoints, and eventually the limitations of the pathology and physiology and anatomy must inevitably draw us closer together

That has happened with mitral stenosis surgery I do not think there is any technical difference in our operations. I have a feeling that that is about what is going to happen in mitral insufficiency procedures Both of us are interested in correcting the annular constriction, a procedure which I feel would be useful in only about one third of the patients, judging by those upon whom we have operated, and the pathologic situations encountered.

The reason I put the emphasis on the improvement in aortic cases was not that I do not think we improved the mitral insufficiency also, but it is so hard to show About the only way one can show improvement in mitral function is by increase in cardiac output. Otherwise one has to take the surgeon's word that there was no more regurgitant jet, or very little, when he got through with his operation. Often there is a residual systolic thrill, and so it is hard to say for some time what improvement is taking place.

We have done only 3 cases of mitral insufficiency by the new method.

As far as aortic insufficiency is concerned, we believe that about half the operative cases are of the annular type, a much larger percentage than in those with the mitral lesion. I think most of those Dr Hufnagel has been operating on are those in whom there is annular dilatation, because he avoids those with significant stenosis The method of putting in the stent involves treating the coexisting stenosis

We took pressure measurements at operation in only two patients, and in both of them there was a differential created between the aorta and the left ventricle—in one case 20 mm. of mercury systolic, and in the other 30 mm. of mercury systolic. We do not think that was excessive, and we think left heart catheterization will indicate that this will be reduced by the loosening of the constriction which does occur in the first 24 hours

We have been impressed by the amount of systolic thrill that one can produce as one tightens this up, but the most important thing is that suddenly the diastolic thrill, which is so palpable over the left ventricular outflow track, is gone. If you put the finger in the left atrium you can feel the septal leaflet of the mitral valve. The thrill is gone, and the impact of aortic closure becomes very palpable. On the following day there is a good, loud auscultatory sound, quite different from what it was preoperatively

You can always check the amount of valvular tightening by a finger in the aorta, which we have done in half of the cases but not in all because lately we thought we could dispense with that particular part of the technique.

AORTIC STENOSIS AND AORTIC INSUFFICIENCY

JOHN H GIBBON, JR (*Philadelphia*)—CHAIRMAN

AORTIC STENOSIS: A POST-MORTEM CINEPHOTOGRAPH STUDY OF VALVE ACTION*

I. K. R. McMillan (*London*)**

In a previous communication[10] we described a method of stud behavior of aortic and pulmonary valves post mortem in an apparatus to simulate the natural conditions of pulsatile flow. In this way a phot record is obtained of the action of normal and abnormal valves and th of surgical procedures can be evaluated.

Aortic valvotomy has been performed in this country and in the States for the last two years.[1,2,3,4,9] The object of this communicat present the information obtained by this method which is relevan selection of cases for this operation

Method. The apparatus described in the previous paper h considerably improved (Fig. 1). Water was found to be the most sat perfusion fluid for photographic purposes. The water is pumped reservoir (fluid tank) to the heart via a solenoid magnetic valve (1 inc eter) which allows a continuous flow of about 25 liters a minute or a p flow of 10–12 liters a minute by the method previously described. After into the ventricle and up through the aortic valve, the water is led t a compressible rubber tube, which in conjunction with an air c (similar in principle to that used in the standard Starling heart-lung ma allows variations in outflow and elastic resistance. The water then ret a rotameter to the reservoir.

In this way the aortic valve can be perfused at a flow equivalent in life. The photography is as previously described. It must be emp that the results recorded are based on studying the working specim cinefilms made from it, and only a few stills taken from them hav used as illustrations

RESULTS

Figure 2 shows one cycle of a normal valve. Thirty aortic valves have been studied Each specimen has

* Reprinted with modifications from the British Heart J

** Mackenzie MacKinnon Research Fellow of the Royal C the Royal College of Surgeons Also assisted by a grant from of St Thomas's Hospital.

PERIPHERAL RESISTANCE
CAMERA
FLOW CONTROL
PRESSURE CONTROL
HEART CHAMBER
FLUID TANK
AORTIC MANOMETER
FLOW METER
MAGNETIC VALVES
VENTRICULAR MANOMETER

Fig. 1 The apparatus used for studying heart valve function.

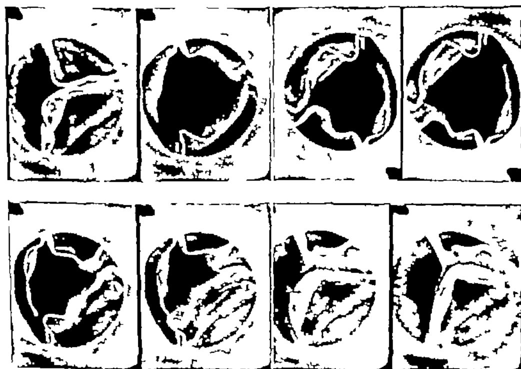


Fig. 2 A cycle of normal aortic valve movements showing the opening and subsequent closure.

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* Reprinted with modifications from the British Heart Journal, *17* 56, 1955

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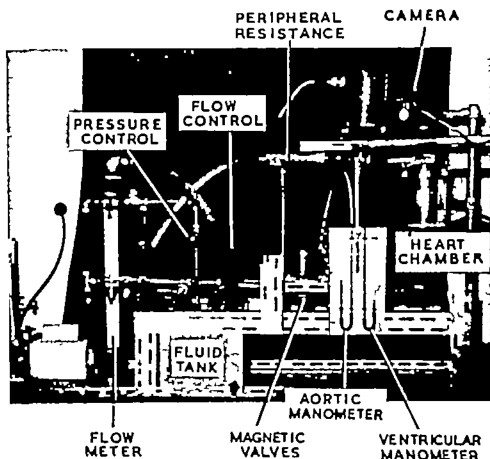


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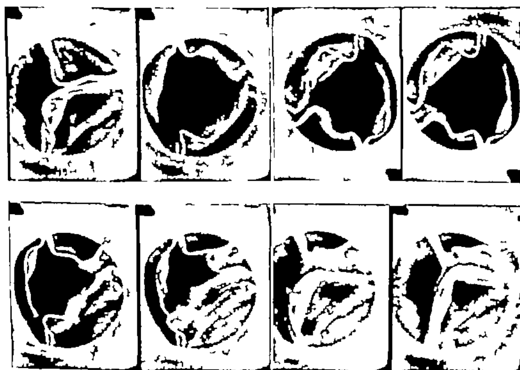


Fig. 2. A cycle of normal aortic valve movements showing the opening and subsequent closure.

machine and, if the data were available, exposed to conditions as near as possible to those obtaining in life. During continuous and pulsatile flow the valves were studied and photographed. In some cases pressures were recorded with a Sanborn electromanometer, above and below the valve, and in a few cases the valve orifice was measured. The first pressure corresponds to the aortic and the second to the left ventricular pressure.

Attempts were made to split the commissures as far as the aortic wall. When the maximum possible split had been obtained, the specimens were studied again Of the 30 specimens studied only 3 were not calcified. The



Fig 3. Peripheral fusion. *a*, Open and *b*, shut



Fig 4 Fibrous fusion with eccentric stenosis *a*, Open and *b*, shut *c* and *d*, Open and shut after post-mortem valvotomy

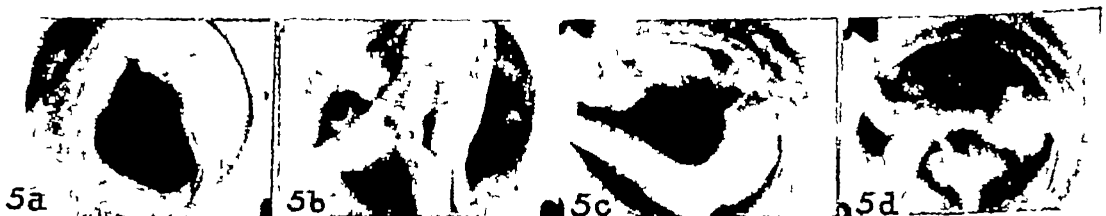


Fig. 5 Fibrous fusion with three commissures fused *a*, Open and *b*, shut *c* and *d*, Open and shut after post-mortem valvotomy.

distribution of calcification varied from small spicules to large masses and the ability to split the commissure depended directly on the amount of calcification present as it was usually densest in the region of the commissure. Of the three fibrous valves, one had early peripheral commissural fusion with little stenosis (Fig. 3), while the second had severe eccentric stenosis (Fig 4), and the third central stenosis with all commissures fused (Fig. 5).

Six specimens were examined after the patient had had an aortic valvotomy in life but had died shortly afterwards.

The shape of the orifice varied in every case, but fell into certain groups.

(1) Early peripheral fusion with good function centrally (3 cases) (Fig 3)
(2) Peripheral calcification not affecting function (1 case) (Fig. 6)
(3) Fusion of one commissure (14 cases) (Figs 7 and 11)
(4) Partial or complete fusion of two commissures (5 cases) (Fig 8)
(5) Partial or complete fusion of three commissures, often with incompetence (5 cases) (Fig 9).
(6) Cone-shaped valve with an ellipsoid orifice at the apex, and no sign of former commissures (2 cases) (Fig 10)



Fig 6 Peripheral calcification, not affecting function. Shut.





Fig. 7 Bicuspid valve, heavily calcified. *a*, Open and *b* shut.









Fig. 8. Aortic stenosis and incompetence with one normal and two fused commissures. *a*, Open, *b*, shut, *c* and *d* after post-mortem valvotomy showing immobility of divided commissure.





Fig. 9 Aortic stenosis and incompetence with three commissures fused (*a*). *b* No improvement after post mortem valvotomy





Fig. 10 Congenital aortic stenosis. *a* Open and *b*, shut.

Types 1 and 2 were presumably degenerative and were accompanied by severe atheroma of the ascending aorta. In Type 3 one quarter were rheumatic and the etiology of the remainder was unknown. Types 4 and 5 usually had an associated mitral valve lesion and were presumed to be rheumatic. The type in some cases depended on the etiology and Type 6 was usually considered to be congenital and known to be in the specimen shown in Fig. 10 This was very difficult to split and was comparable to the cone-shaped congenital pulmonary valve.

RESULTS OF VALVOTOMY POST MORTEM

In the first fibrous case (Fig. 3) the lesion was not clinically detectable and no treatment would have been required, in the second (Fig. 4) one com-

missure split easily with the finger and the other with the knife, and in third (Fig. 5) they split easily with the Bailey dilator. If these last two c could have been accurately diagnosed in life, a very good surgical re would have been possible as in the first the flow through the valve increa from 1.9 to 5.5 liters after valvotomy and the pressure difference across valve decreased from 70 to 20 mm. Hg.

The results in the calcified valves were on the whole disappointing, a most cases there was considerable calcification in the fused commissures even if a split was made with a knife, the rigidity of the valves was such t neither movement nor flow was greatly increased (Fig. 8). If an elec valvotomy could be performed on the commissure mainly affected a g result could be achieved (Fig. 11).



Fig 11. Bicuspid aortic stenosis *a*, Open and *b*, shut *c*, Shows good result selective post-mortem valvotomy



Fig 12 Bicuspid aortic stenosis after valvotomy in life (split at lower right ha corner) *a*, Open and *b*, shut

Figure 12 shows the effect of dilating a bicuspid valve in life. Here t fused commissure has split to the aortic wall and some mobility has be restored.

Insertion of a dilator produced either no commissurotomy, or splitting one or two of the commissures, but in no instance was a tri-radiate sp obtained by dilation. Selective splitting of the third commissure was or possible with the knife, a method as yet impracticable in life. Figure shows that with the Bailey dilator[1] results can be very disappointing, as th specimen had a valvotomy in life It also shows the Bailey dilator in t same calcified valve performing valvotomy post mortem (*c* and *d*) and t results of its action after full opening (*e* and *f*). In no case was a calcifi valve split except in the region of the commissure. But if one commissu was normal and the rim of the cusp soft, this latter was seen to tear in sever cases rather than the calcified area split (indicated by the arrow in Fig. 13*b*

Figure 5 shows a fibrous valve, and an optimal result after post-morte valvotomy with a Bailey dilator. Figure 8 shows calcification on one si

TABLE I RESULTS OF AORTIC VALVOTOMY

No	Valve Type	Calcification	Valvotomy	Result
1	Bicuspid	++	1 commissure	Poor
2	"	+++		No record
3	"	+++		Some improvement
4	"	+	—	
5	"	++	1 commissure	No improvement
6	(Fig. 8)	+++		Slight improvement
7	(Fig 11)	+++		Good
8	(Fig 7)	++	—	
9	"	+++	—	
10	Congenital bicuspid	+++	1 commissure	Much improvement
11	Bicuspid	++	—	
12	Round hole (Fig. 16)	+++	—	
13		+++	—	
14	(Fig 9)	+++	3 commissures	No improvement, increased incompetence
15		++	—	
16		++	—	
17	1 commissure normal—2 partly fused	+++	—	
18		++	(*a*) 1 commissure split (*b*) 2 commissures split	Slight improvement No further improvement
19		++	—	
20	2 commissures completely fused, 1 normal (Fig 4)	0	2 commissures	Much improvement
21	Partial fusion 3 commissures	+	—	(Fig. 3)
22	Partial fusion 3 commissures and incompetence	0	—	
23	Peripheral calcification but no fusion (Fig 6)	+	—	
24	1 commissure completely fused, 2 partly (Fig. 5)	0	2 commissures split with Bailey dilator	Much improvement
			Commissurotomy during life	
25	Bicuspid	++	in vivo	No split
26	(Fig. 12)	++		Small split
27		+++	"	Fair
28	Congenital cone shape	+	"	Minimal split
29	" (Fig 10)	++	"	" "
30	2 commissures completely fused, 1 normal (Fig. 13)	++	"	Little improvement

and mobility on the other. Here splitting of one commissure helped a little but as the calcification occupied half of the fused cusps, attempted dilatation through the pliable part merely pushed the solid part laterally without splitting any further.

DISCUSSION

Leonardo da Vinci[8] first demonstrated that the normal aortic valve opened to give a triangular orifice and this has been amply confirmed (Fig. 2). It follows, therefore, that the normal orifice is considerably less than the cross-sectional area of the aorta: this is approximately 5.3 sq. cm. (Quain) and the calculated area of the triangle is 2.6–3.5 sq. cm. approximately.

This in practice means that as long as the free borders of the cusps are mobile and shut well, there may be considerable calcification in the peripheral parts of the cusps and walls of the sinuses of Valsalva without limitation of flow in the valve area (Fig. 6). It may be difficult to distinguish the various sites of calcification on radiologic evidence alone.

Some fusion of the peripheral parts of the commissures can occur without causing symptoms (Fig. 3). The maximum aortic orifice is much greater than the normal requirement and this combined with the compensatory hypertrophy of the left ventricle provides an explanation for the advanced pathologic change often seen post mortem compared with a relatively short disability. It is intended to present data on the critical orifice size and their pressure relationships elsewhere (Gorlin[6] et al.).

Experience with mitral valvotomy suggests that the bivalve structure of the mitral valve can be split with the finger or knife even when calcified (Figs. 14 and 15). But the surgeon has the inestimable advantage of being able to feel the valve and know where the commissures should be, whereas aortic valvotomy at present has to be done blindly by an instrument, so that chance plays a bigger part in deciding the site of the valvotomy. The aortic approach is an advance but the finger can only guide the instrument to the orifice and not select the commissure to be split (Brock[4]; Bailey[1]) As mentioned previously valvotomy usually splits the weakest commissure and rarely more than two commissures. In some cases the valve may have been a congenital bicuspid type with a fused raphe representing the third commissure, but even where three commissures were present the third was very difficult to split.

The ventricular approach, however, is the one most commonly used Attempted blind dilation of a calcified ring from some distance above the valve may lead to a separation of the aortic wall from the calcified valve which may have dire results as was seen in one case. Another disturbing factor was the roughness of the valve surface, even before splitting, with fibrin and calcified particles only loosely attached. This is much worse after splitting in all except the fibrous valves and makes only too obvious the possibility of peripheral systemic embolism as a result of valvotomy (Fig 16).

When one commissure is unaffected a relatively normal cusp may be torn and aortic incompetence result. An early degree of this is shown in Fig. 13*b* and is marked by an arrow This hazard can only be eliminated by direct



Fig. 13 Calcific aortic stenosis after valvotomy in life. *a*, Open and *b* shut. *c* and *d* With Bailey dilator in situ open and shut. *e* and *f* Show result of post mortem valvotomy with Bailey dilator



Fig. 14. Normal mitral valve seen from left atrium with aortic cusp on right. *a*, Open and *b*, shut.



Fig. 15 Calcific mitral stenosis with aortic cusp on right. *a*, Open, *b* shut *c* and *d*, after post-mortem valvotomy showing optimal result.



Fig. 16 Aortic stenosis and incompetence with rough edges of the valve. *a* and *b* Shut and open.

palpation or inspection of the valve. Fig. 13*f* shows how the damage is increased after repeated post-mortem dilatation of the same specimen.

The chances of a recurrence of the stenosis by fusion of the divided commissure would appear to be high owing to their rough edges and frequent limitation of mobility of the cusps due to calcification, even after splitting (e.g Fig 8) The best results would be anticipated in fibrous valves without gross calcification. An accurate method of determining the presence or ab-

sence of calcification and of its distribution is urgently needed to improve the selection of cases for operation. In patients with much calcification, exploration is worth while if the circumstances justify the risk, as a proportion of such patients may have a commissure amenable to splitting, or the calcification may be outside the critical orifice area

The orifice size, measured from films taken of the valve working under conditions simulating those in life, was smaller than that obtained by direct measurement with the finger. This was due to the fact that the examining finger post mortem can exert a relatively enormous pressure compared to that produced by the contracting ventricle in life This would account for what appears to be a good split producing a poor functional result and is correlated exactly with the degree of rigidity of the valve and particularly with calcification

The method as described in this paper gives very useful information in determining valve action and the effect of surgical procedures on the valves, and by cinematography allows permanent records to be made. This study has been devoted purely to valve function and no attempt has been made to analyze the differences due to differing etiology.

CONCLUSIONS

Thirty stenosed aortic valves were studied post mortem in an artificial perfusion system Of these 27 showed various degrees of calcification.

The effect of post-mortem valvotomy was studied in 25 specimens, and the results of valvotomy in life in a further 6 specimens.

Many valves after valvotomy were still relatively immobile. Usually it was only possible to split one or two commissures, even with tri-radiate dilators. Unlike mitral valvotomy, large increases of valve area were not easily obtained, except in the uncalcified specimens

When two commissures were fused, forcible dilatation in some instances damaged the remaining mobile cusp This could lead to the production of incompetence in life

The uncalcified stenosed aortic valve is the most suitable for aortic valvotomy as it can be easily divided

A method of direct inspection of the valves is urgently needed to assess operability, as this cannot be done accurately by existing clinical methods.

The presence of gross calcification seriously militates against a successful functional operative result, but the existing methods of determining the degree and distribution of calcification in life are too imprecise to forbid operation if the circumstances justify the risk

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Question Does Dr McMillan have any information on the mitral valve? Did he study it by the same method?

Answer The answer is 'Yes We have the same sort of series of various types of mitral stenosis and mitral incompetence in another movie, but I do not have it here.

I would emphasize that this method is less applicable to the mitral valve, obviously, because, as was discussed previously, the contraction of the atrial ring as shown is something I cannot copy I can merely show the valve action. I do not think that is so important to the aortic valve because the ring does not move in the way the mitral ring does, and at the worst it would dilate to a maximum and then stay there.

Question I would like to ask Dr McMillan whether he has done any longevity tests with any type of flap grafts, perhaps using blood for a period of support and making it work continuously All the flaps, of course, would not have any blood supply other than that coming from the oxygenated blood in the circuit.

Answer We have done it around the descending aorta in dogs, but the trouble was the amount of clot that forms behind the valve, and I think this is where the Hufnagel valve is superior at the moment. It can be made to work quite simply, but it works for only four or five hours We haven t gotten over that problem at the moment.

As regards those particular flaps, they were worked for three or four hours, which I agree is a very poor control, but at least working at speeds up to 110 to 120 they did not fly to bits

SURGICAL TREATMENT OF AORTIC STENOSIS

CHARLES P. BAILEY (*Philadelphia*)

We have been interested in and working upon the problem of aortic stenosis for almost five years, and have had significant success for just about



Fig 1. Rheumatic aortic stenosis with the two posterior commissures well parted, the anterior commissure was open, but rather rigid (From Larzelere and Bailey Journal of Thoracic Surgery, vol 26, 1953)

three years. The original operative procedure has undergone considerable modification, and I will try to show this by slides.

As Dr. McMillan has indicated, the most significant lesion in aortic stenosis is one of fusion of the commissures, which is seen beginning in Fig. 1 Then, a point which he did not make is that calcification ordinarily appears first at the commissures and only secondarily in most instances in the sinuses of Valsalva. However, that phenomenon is not by any means constant. Gradually the orifice is reduced by this process to a much smaller one, which in this specimen you see is a nice central triangular one, and the fusion of the commissures is evident, as also is calcification both there and in the sinuses. However, he has also indicated that you may have bizarre and eccentric types of valve orifices.

Figure 2 is an extreme example of a calcified valve. It has come to resemble a clam shell, and one so dense that even in the autopsy room it could hardly be separated with the metal instrument.

Obviously one can do very little for patients with the most extreme examples of this pathology On the other hand, if there is some residual valve function, if there is some residual valve flexibility, or if the commissures can be opened all the way to the annulus, which may still retain some mobility, one can hope to do some good for the patient.



Fig. 2. Calcified aortic valve. (Courtesy of Dr George Geckeler, Philadelphia.)

In one of our patients the preoperative systolic pressure in the left ventricle was well over 200, while that in the aorta and brachial artery was of the order of 130 Such a gradient across the aortic valve is pathognomonic of severe aortic stenosis

This same patient was operated on, and at surgery a catheter was threaded through the left auricular appendage, through the mitral valve, into the left ventricle and into the aorta, and as it was drawn back we obtained pressures which showed a much smaller differential than were obtained on the same patient without his chest opened.

I think that is a very important observation, indicating that pressures taken during surgery must be considered unreliable and without the same signifi-

cance that they have when obtained by left heart catheterization either by the method of Bjork or by the transbronchial route

Figure 3 shows the instrument Dr McMillan referred to It has three dilating blades and a swivel mechanism at the junction of the head with the shaft so that it can rotate freely to adjust to a triangular aperture and, hence, to apply pressure properly against the fused commissures. It has been designed and is produced by Major J Shearman Donaldson of Chatham, N. J

However, as Dr. McMillan has shown, you don't always separate even two commissures You may part only one. If it is one of these badly deformed, calcific or bicuspid valves, you may produce a bizarre fracture of the valve, and cause regurgitation or even death of the patient.

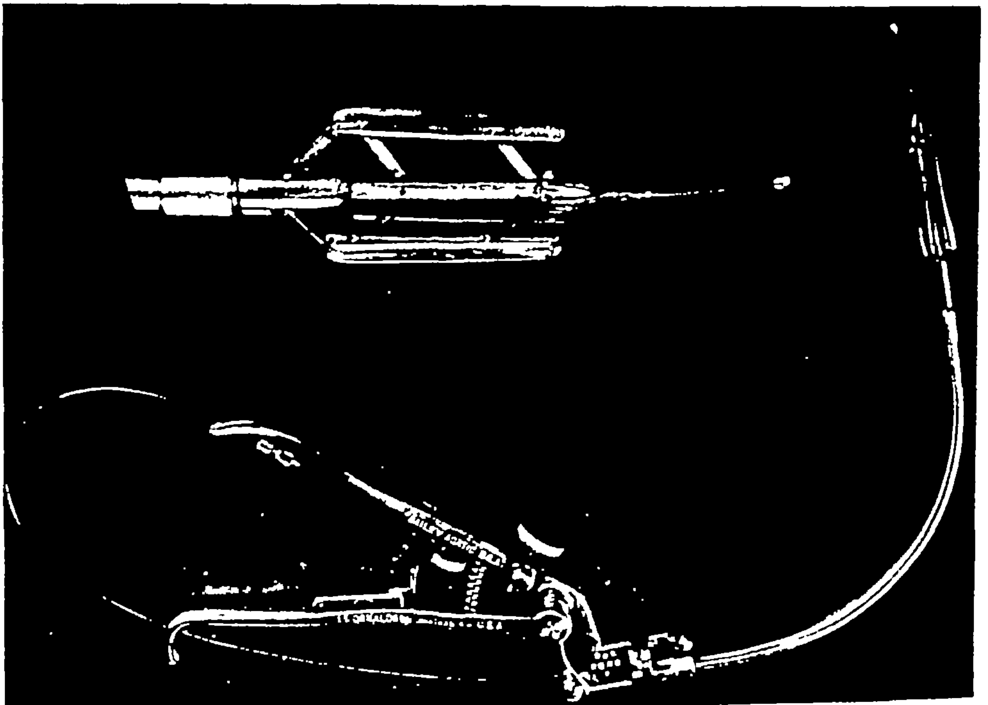


Fig 3 Bailey aortic dilator

The transventricular technique has been presented previously A small puncture is made in the left ventricle (Fig 4) The guide wire is inserted and directed upward along the septum It goes through the aortic valve easily. You can squeeze the root of the aorta with the fingers of the left hand, and when you feel it to have passed through you know you will not create a false passage

The incision in the ventricle is enlarged slightly, the closed instrument is introduced and is passed up along the wire through the valve until you can feel half of it in the aorta (Fig 5) Then you squeeze the handles, and the blades should split one or more commissures of a triangular opening quite satisfactorily if it can be split.

Altogether we have done 211 operations for aortic stenosis via the transventricular route, using this instrument in all but 16 patients. In order to evaluate properly the results of that effort, we have broken down our cases into various groups.

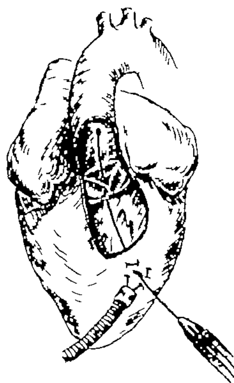


Fig 4



Fig 5

Fig. 4 Guide wire advanced through the left ventricular outflow tract and stenotic aortic valve and into the aorta.

Fig. 5 Triradiate bars of the aortic dilator expanded, separating the commissures of the aortic valve. (Both figures from Bailey and others, Journal of International College of Surgeons Vol. 20 1953)

Table 1 includes the group of cases that had either pure aortic stenosis or a completely adynamic aortic insufficiency with it and a normal mitral valve. In 68 patients grouped as shown according to the American Heart classification we had 19 hospital deaths, a mortality of 28 per cent. That seemed unreasonably high to us, even though several operators had performed these operations It was a beginning series, and some patients may have been lost due to our inexperience.

During this same period there were 87 patients with essentially pure aortic stenosis and pure mitral stenosis, both valves being operated upon at the

TABLE 1 TRANSVENTRICULAR APPROACH FOR AORTIC COMMISSUROTOMY SHOWING EARLY OPERATIVE RESULTS

Type of Lesion	*Number of Cases*	*Number of Operative Deaths*	*Operative Mortality* %
Aortic stenosis	37	9	24.2
Aortic stenosis with insignificant aortic insufficiency	31	10	32
Total	68	19	28

TABLE 2 OPERATIVE RESULTS IN COMBINED SIMULTANEOUS AORTIC AND MITRAL COMMISSUROTOMY IN PATIENTS WITH AORTIC AND MITRAL VALVULAR DISEASE (TRANSVENTRICULAR AND LEFT AURICULAR APPROACHES)

Type of Lesion	*Number of Cases*	*Number of Operative Deaths*	*Mortality %*
AS plus MS	22	1	4 5
AS plus MSmi	10	2	20
ASai MS	24	4	16 7
ASai MSmi	31	9	29
Total	87	16	18 4

i = mild insufficiency

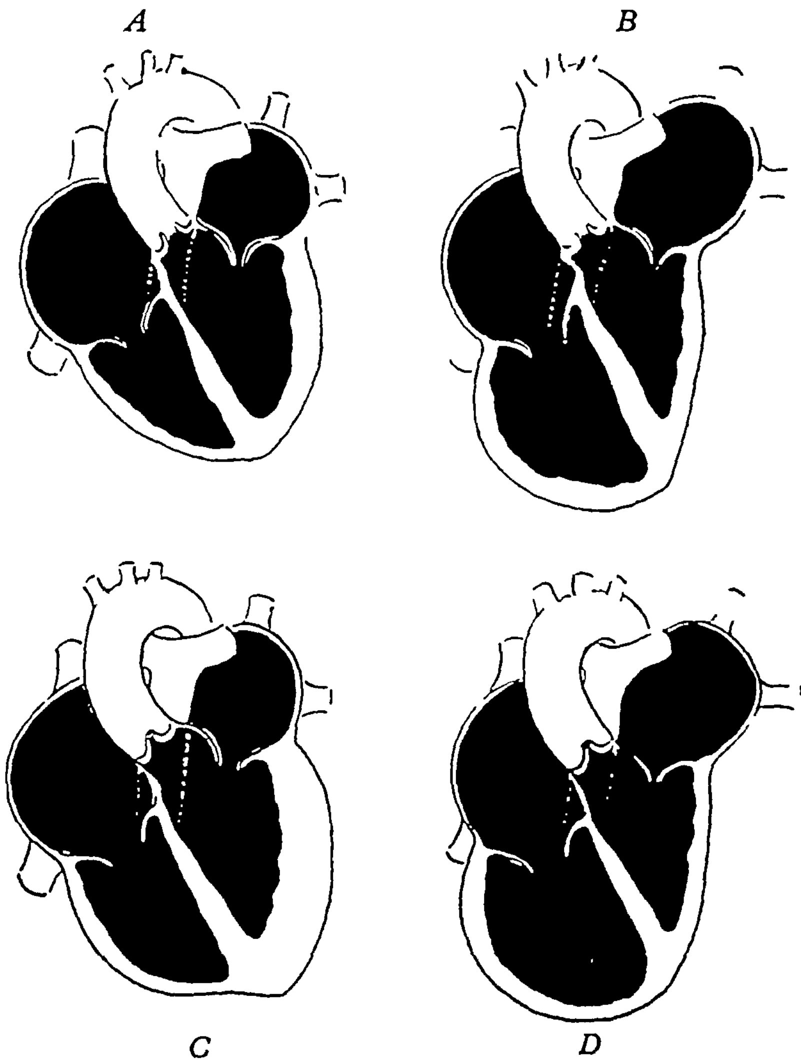


Fig. 6. The effects of valve lesions on the cardiac chambers. *A*, Normal heart. *B*, Mitral stenosis with small left ventricle *C*, Aortic stenosis with enlarged left ventricle, and possibly dilated right ventricle *D*, Both mitral and aortic stenosis

same time, the mitral valve through the left auricular appendage and the aortic by the transventricular route (Table 2). We left out of consideration all those with significant insufficiency of either valve. There were 16 deaths

in this group, or a mortality of 18 4 per cent. This was considered to be significantly lower than the other, especially when you consider that two valves were being operated on instead of one. That observation led to considerable speculation.

Figure 6 contains diagrams illustrating the effects of various valve lesions upon the cardiac chambers—*A* being the normal heart. *B* shows the heart with mitral stenosis showing a small left ventricle. *C* is the heart with pure aortic stenosis and a normal mitral valve with a big left ventricle, maybe also a dilated right ventricle because of backward failure. *D*, indicating both valves stenotic, shows that such a heart looks almost exactly the same as one with *isolated mitral stenosis* The left ventricle is small because the trickle of blood that goes through the mitral valve can easily be expelled through the aortic. Hence, the combined lesion avoids the worst hydrodynamic effects of aortic stenosis

We decided that there generally were two causes of death during transventricular aortic commissurotomy (Table 3) One was related to the left ventricular stimulation or approach, because one of the causes of death was

TABLE 3 CAUSES OF DEATH IN PATIENTS OPERATED FOR AORTIC STENOSIS BY TRANSVENTRICULAR TECHNIQUE

Cause of Death	*Number of Deaths*
Failure, unclassified	1
Cardiac arrest	5
Ventricular fibrillation	5
Hemorrhage, uncontrolled	2
Cerebral embolism	3
Hypotension (after leaving operating room)	1
Hypotension (in operating room)	1
Cardiac arrest / Ventricular fibrillation / Laceration of coronary artery	1
	19

bleeding from incisions in the left ventricle that could not be sutured because of a peculiar softness of the left ventricular myocardium, and another cause of death was the creation inadvertently of aortic insufficiency We concluded that it would be necessary to perform this operation in the same manner as commissurotomy for mitral stenosis—by the use of digital guidance.

Implementation of this concept was long and tedious, with a lot of animal experimentation first. The way this has been worked out requires one to apply a curved Potts clamp or a modified Satinsky clamp to the *ascending* aorta, pinching up a portion of its lumen. An incision is made longitudinally in the excluded portion.

We have tried various substances to make a pouch or an appendage on the aorta. We have used plastic, latex, cloth, and we still like pericardium better than anything else. We use homograft pericardium preserved according to the original method of Meeker and Gross. The pericardium is thawed out. A selected portion is encircled with a purse-string suture and a central longi-

tudinal opening is made. The lips of the central incision are sutured in an overlapping or imbricating fashion to the cut edges of the aortic wall This suture technique is very important, because the aortico-pericardial junction always leaks if any other method is used. Plastic pouches are already more

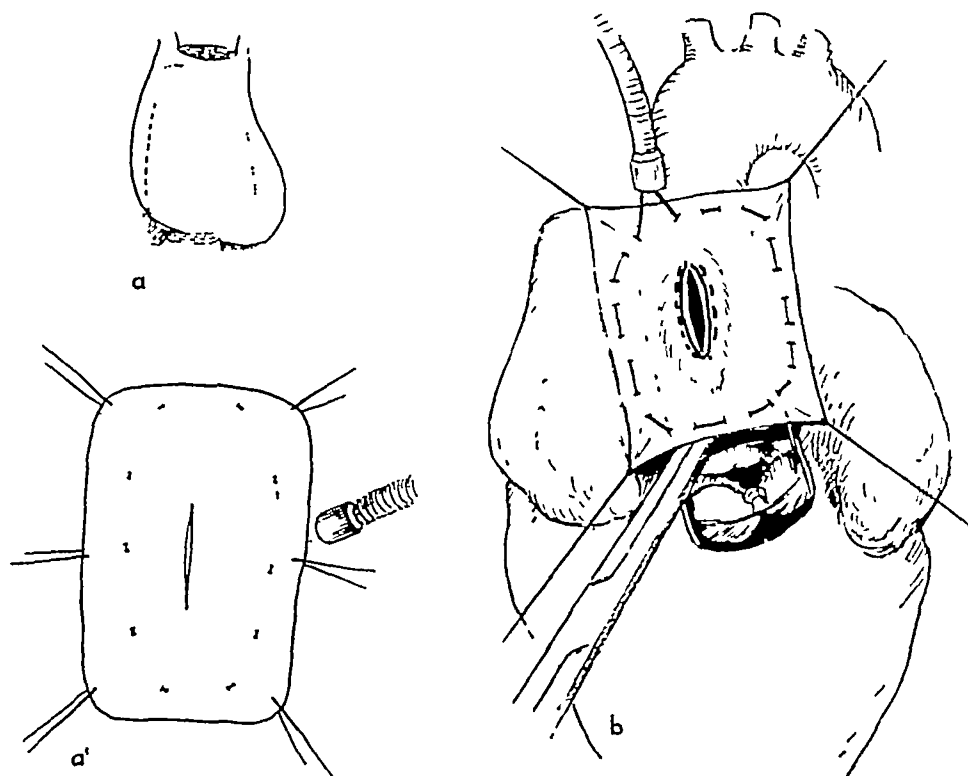


Fig 7. Preparation of the pericardial pouch. *a*, Resecting the pericardial patch *a'*, An incision at least 4 cm in length is made longitudinally in the patch. *b*, Suturing the patch to the aortic wall

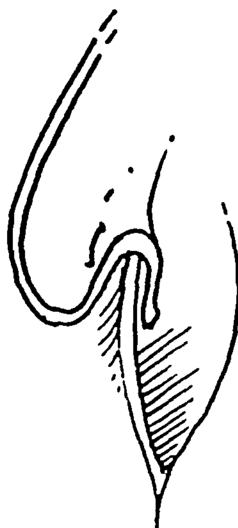


Fig 8. Suture technique for aortic pouch

or less shaped, and they are hard to sew on properly because one can't get to both sides.

In Figure 8 the suture technique is demonstrated. We employ a mattress suture, back and forth, through two layers of enfolding pericardium and one intervening layer of aortic wall.

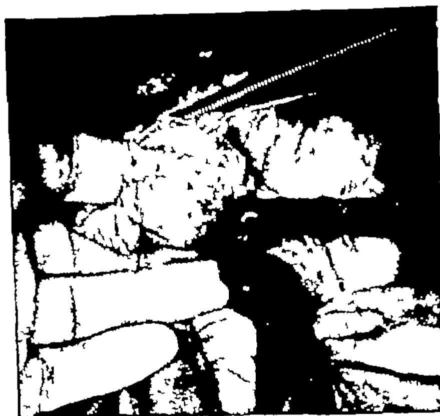


Fig. 9 Photograph of ungloved left index finger in distended pouch just before release of aortic clamp (From Bailey Surgery of the Heart. Courtesy of Lea & Febiger)

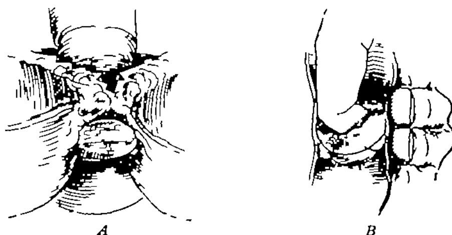


Fig. 10 *A* Hooking finger tip under a fused commissure and making upward disruptive traction. *B* Same plus aid of external counterpressure. (From Bailey Surgery of the Heart. Courtesy of Lea & Febiger)

The purse-string is tightened, the sac is filled with saline (Fig 9) As you see we use a bare finger technique. Usually we just cut the rubber finger off the glove instead of having the whole hand bare, but in obtaining this photograph we wanted to demonstrate that we do use a bare finger technique. I don t think you can do a proper job with a glove on.

Then you release the clamp and insert the finger down into the valve, using the same kind of maneuvers as you do in attempting to split a stenotic

mitral valve (Fig. 10). In our series of 61 patients operated on by this technique, over 40 per cent of the valves have been split by the finger alone—almost the same percentage as in the mitral valve. You can use the other hand on the outside of the aorta for counterpressure.

Strangely enough, the fibrotic type of valve may resist the finger more tenaciously than the calcific type, which often tends to split in the line of commissure pretty well.

Obviously we don't split all three commissures in many patients. If we can end up with one commissure separated to the wall of the aorta, and another one two thirds of the way, we consider that is a good result.

Hydrodynamically, aortic stenosis is not too significant until the aperture is reduced to one quarter of its normal. If you can open the passage more

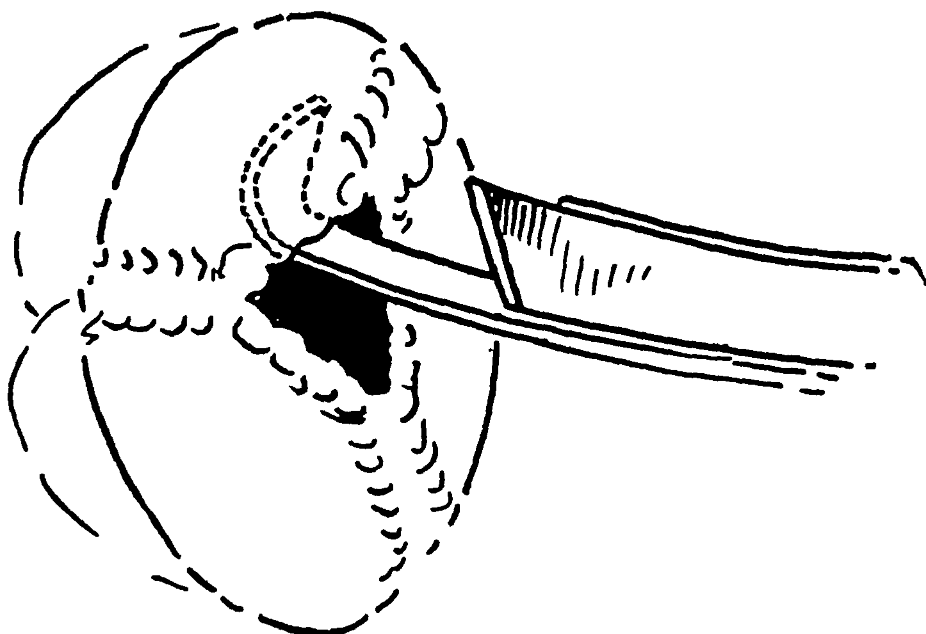


Fig 11. Shearing through a resistant commissure with one of the mitral valve guillotines (From Bailey Surgery of the Heart Courtesy of Lea & Febiger.)

than 25 per cent of normal, the heart, which is already adjusted to a serious degree of aortic stenosis, will react as though it had been relieved of its strain

In resistant commissures, as first suggested by William Swann, you can use a guillotine knife such as those we use for mitral commissurotomy (Fig. 11). If you cut exactly in the right line you are all right. If you cut a little off the line, I shudder to think what would happen. I don't think so far we have cut off the proper line, or at least not far enough off so that the incised edges of the stiff valve have tended to turn down.

More often we use this dilator prepared by the George Pilling Co. of Philadelphia, which has been devised to be worn on the finger (Fig. 12). It has only two blades which open at a 110 degree angle.

Figure 13 is an actual photograph showing the dilator on the finger, going into the pouch and extending down into the aorta in the region of the valve. There is not a great amount of bleeding taking place. Part of the excellent

hemostatic control is due to the purse-string; part of it is due to this gauze which has been wrapped around the lower part of the pouch at this point. That is a very valuable additional precaution.

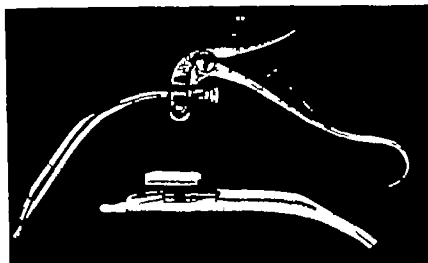


Fig. 12. Two-bladed retrograde aortic dilator Top, Closed instrument. Bottom, Dilating blades expanded. (From Bailey· Surgery of the Heart. Courtesy of Lea & Febiger)



Fig. 13 Retrograde dilator advanced along the finger as the purse-string suture is tightened and the gauze is released. (From Bailey· Surgery of the Heart. Courtesy of Lea & Febiger)

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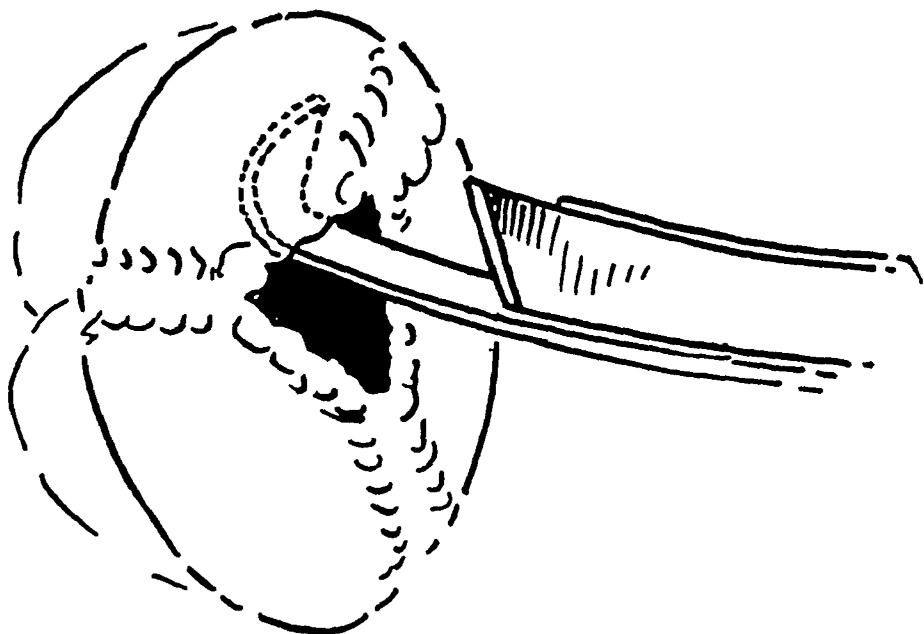


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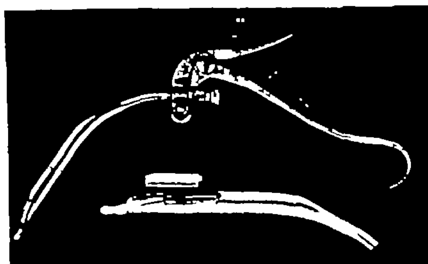


Fig. 12. Two-bladed retrograde aortic dilator Top, Closed instrument. Bottom Dilating blades expanded. (From Bailey Surgery of the Heart. Courtesy of Lea & Febiger)



Fig. 13 Retrograde dilator advanced along the finger as the purse-string suture is tightened and the gauze is released. (From Bailey Surgery of the Heart. Courtesy of Lea & Febiger)

The finger guides the instrument down toward the aortic orifice (Fig 14) You can always find it because blood rushes up out of it with each heart beat. Just follow the jet with your fingertip until you come to the diminu-

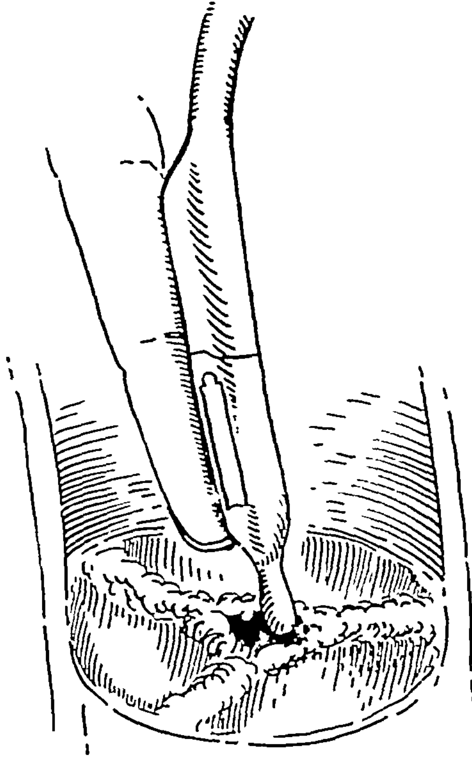


Fig 14 Finger guides the dilator tip into the valve orifice by following the systolic gush of blood emerging from the left ventricle (From Bailey Surgery of the Heart Courtesy of Lea & Febiger)

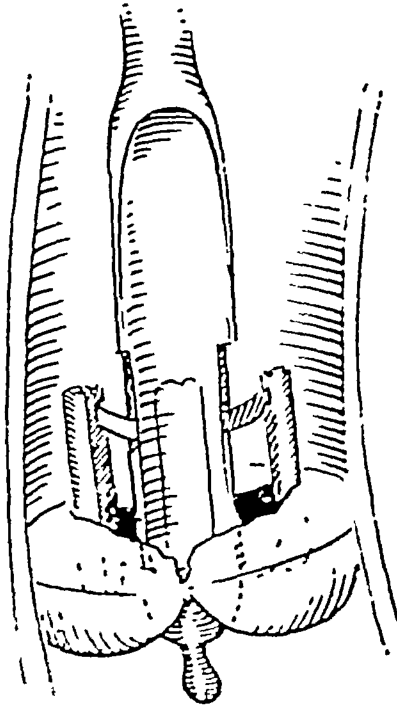


Fig 15 Retrograde dilator in place with blades opened

tive opening. Of course you already will have tried to open it with the finger The tapering tip of the instrument is guided into the opening Then you can push vigorously, and the sloping shoulder will follow through the valve.

After the instrument is placed properly the handle is squeezed by the other hand, the blades spring apart, and two of the commissures are attacked simultaneously (Fig 15) *Actually there is three-point* pressure because the instrument is bulky, more so than we like. Thus there is some pressure applied in three directions

If the third commissure is to be separated, rotate the instrument 120 degrees and open the blades again (Fig 16)

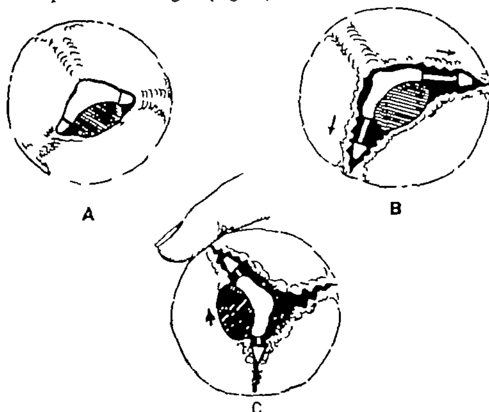


Fig. 16 Retrograde aortic instrumental commissurotomy guided and limited by digital palpation. *A* Dilating edges of instrument applied to left and right posterolateral commissures. *B* By expanding the dilating bars these commissures are separated. *C* After rotating the instrument 120 degrees (clockwise direction) the anterior commissure may be split with the help of external counterpressure. (From Bailey Surgery of the Heart. Courtesy of Lea & Febiger)

Since we adopted this routine we have operated on 47 patients with essentially pure aortic stenosis and very little insufficiency (Table 4) As you can see, we have lost 7 patients in that group—a mortality of 14 7 per cent, less than half of that encountered with the transventricular approach.

At the same time we have operated on 14 patients from above *with combined* aortic and mitral stenosis, and we have had only one death (Table 5) Thus we have had 8 deaths in 61 patients operated *on by the transaortic* approach. The over all mortality has been and should be less than 10 per cent.

Figure 17 *shows the aortic pressure in a patient* operated on in this manner, after surgery We haven t quite abolished all of the transaortic differential,

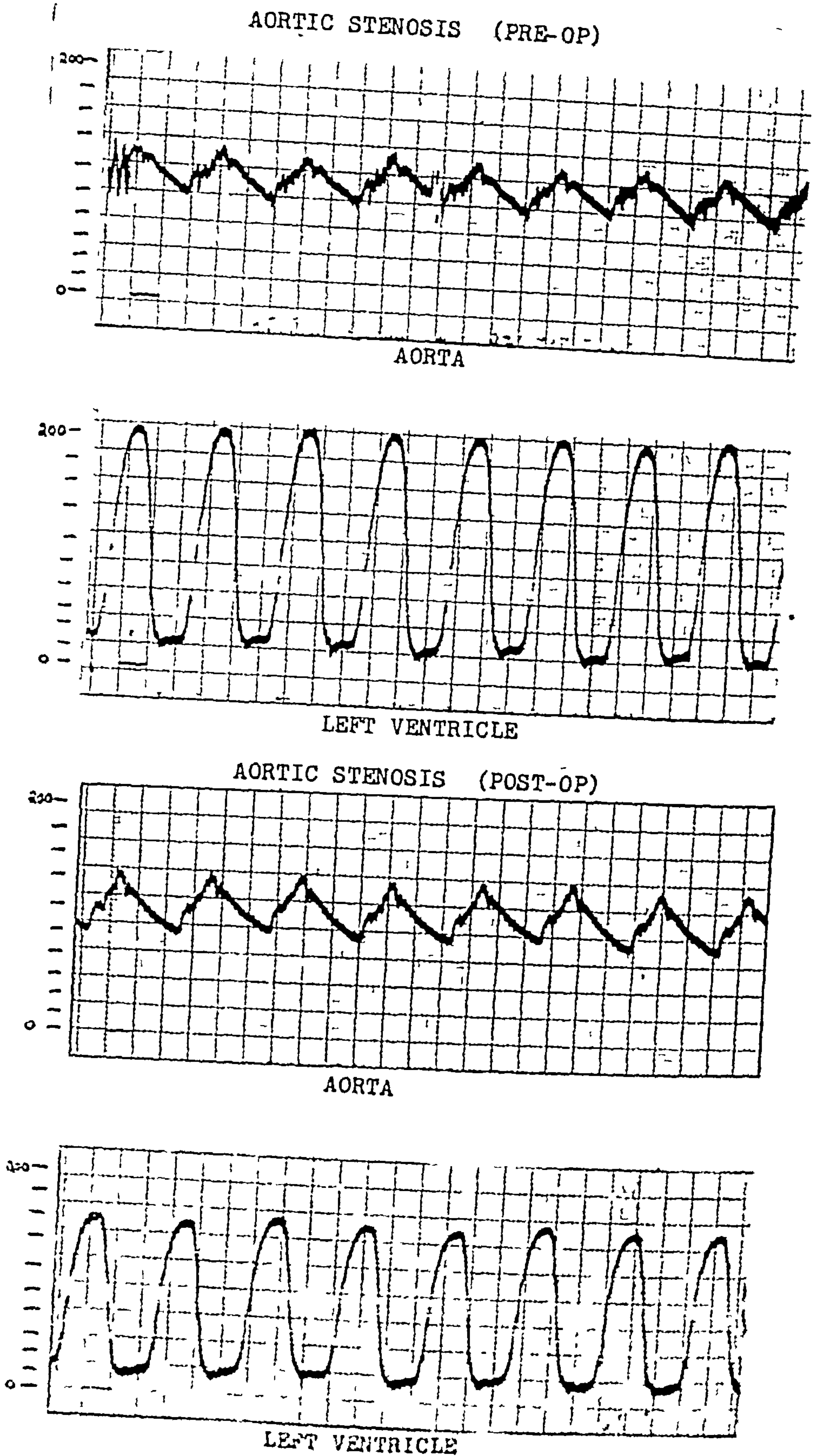


Fig 17 Preoperative and postoperative direct aortic and left ventricular pressure tracings determined at surgery showing abolition of differential by relief of aortic obstruction. (From Bailey Surgery of the Heart Courtesy of Lea & Febiger)

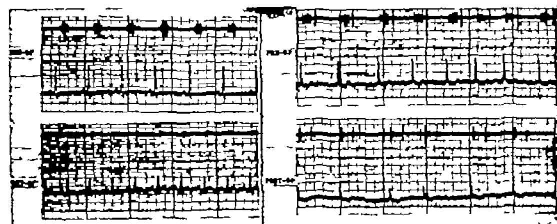


Fig. 18 Preoperative and postoperative phonocardiograms in severe aortic stenosis

TABLE 4. RESULTS OF SURGERY UPON 47 PATIENTS OPERATED UPON FOR AORTIC STENOSIS BY THE TRANSAORTIC APPROACH

American Heart Classification	*Number of Cases*	*Operative Deaths*	*Per Cent Mortality*
I	2	0	0%
II	12	1	8.3%
III	30	6	20%
IV	3	0	0
Total	47	7	14.7%

TABLE 5 OPERATIVE MORTALITY IN TRANSAORTIC APPROACH AND RIGHT-SIDED MITRAL COMMISSUROTOMIES IN COMBINED MITRAL AND AORTIC STENOSIS

American Heart Classification	*Number of Cases*	*Operative Deaths*	*Per Cent Mortality*
I	0	0	0
II	5	0	0
III	9	1	11 1%
IV	0	0	0
Total	14	1	7 1%

but we have reduced it very materially If we get a good result, the brachial artery tracing becomes essentially normal postoperatively

Figure 18 includes phonocardiograms in a patient with severe aortic stenosis, preoperatively and then postoperatively Postoperatively there is a good aortic second sound after surgery that was absent before. There is much less systolic murmur as indicated by the graph.

After considerable experience with this technique I am convinced that the logical approach to the stenotic aortic valve is from above. Even if there *should* be associated mitral stenosis, one can operate from the right side for the

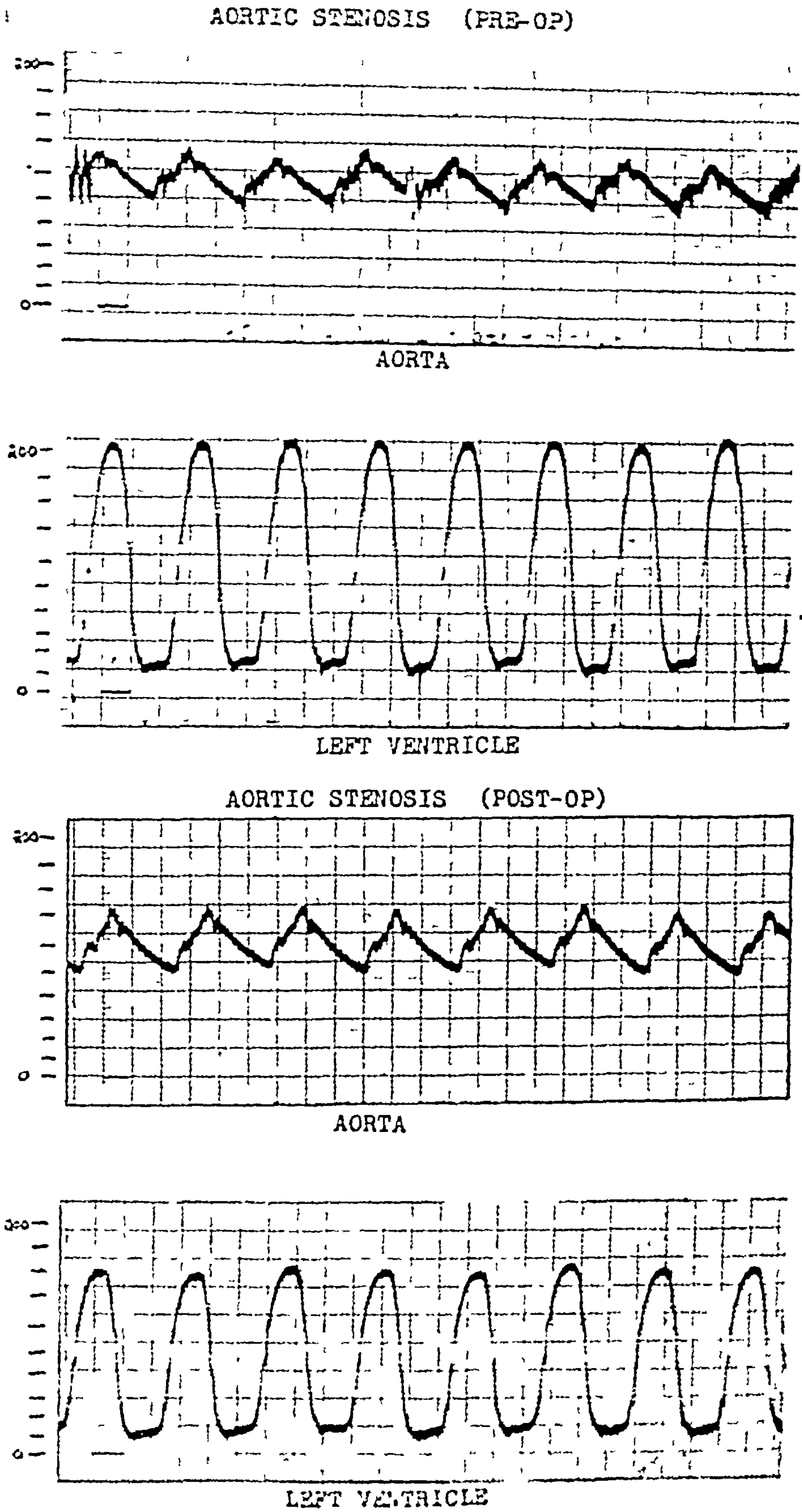


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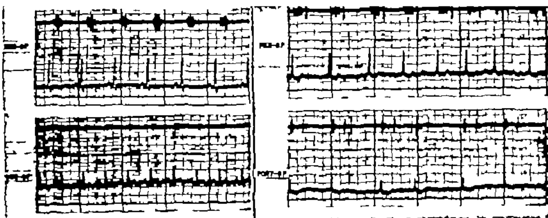


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After considerable experience with this technique I am convinced that the logical approach to the stenotic aortic valve is from above. Even if there should be associated mitral stenosis, one can operate from the right side for the

mitral valve separating the posterior portion of the interatrial septum, splitting it according to the method that Sondergaard has shown so beautifully (Fig. 16, p. 243) This is a very satisfactory way to perform a mitral commissurotomy.

DISCUSSION

William H. Muller, Jr. (*Charlottesville, Virginia*)

This was a most interesting discussion by Dr. Bailey, and I would like to congratulate him upon the contributions he has made in the development of procedures to treat this difficult deformity.



Fig. 1. Valvulotome, (*A*) closed and (*B*) opened

Certainly the problem of treating valvular aortic stenosis is far greater than that presented by mitral stenosis or pulmonary stenosis, for many reasons which I shall not go into now.

One of the chief problems which one encounters, particularly in the older patient with aortic stenosis, is a very low left ventricular reserve, and a markedly calcified and rolled-up valve, as we have seen demonstrated by Dr.

McMillan. Because of this low left ventricular reserve, we have tried to do the operation of shortest duration and one associated with minimal trauma. Therefore, we have used the transventricular approach in a relatively small series of 37 patients

Figure 1 shows the instrument we have used. It is similar to Dr Bailey's It is considerably smaller, however, and for that reason we have had no difficulty, after our initial experiences, in the control of bleeding from the hypertensive left ventricle.

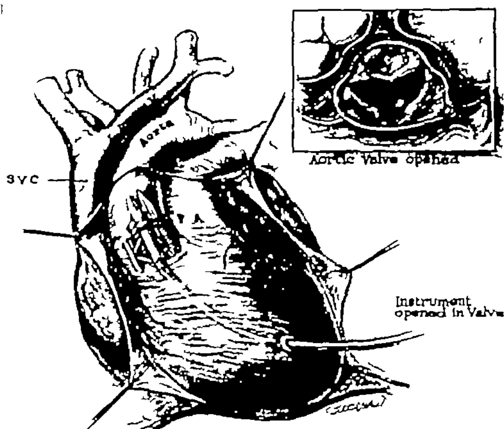


Fig 2. Valvulotome in place in left ventricle Insert shows opening of aortic valve.

Figure 2 demonstrates the valvulotome in position in the left ventricle, and shows what we would like to achieve in the insert.

We feel it is very important to measure pressures in the left ventricle and aorta on either side of the valve. As was demonstrated by Dr McMillan, often one will not achieve an adequate opening on the first attempt. However, pressure measurements will indicate whether or not the obstruction has been relieved. If not, the valvulotome can be reinserted and the valve opened further

Twenty-six of our patients had aortic stenosis and some degree of insufficiency Ten had mitral and aortic stenosis One had coarctation of the aorta associated with the deformity

There were 10 operative deaths Seven had aortic valvular disease alone and 3 had both aortic and mitral valvular disease. This is approximately 27 per cent which compares with Dr Bailey's transventricular series Sixteen of

ment is much smaller than the original dilator used for this surgery, but it expands a few millimeters further than the medium size dilator available. Incidentally, these pieces or heads are attachments which can be put on the original dilator The handle mechanism of the original dilator is in the sur-

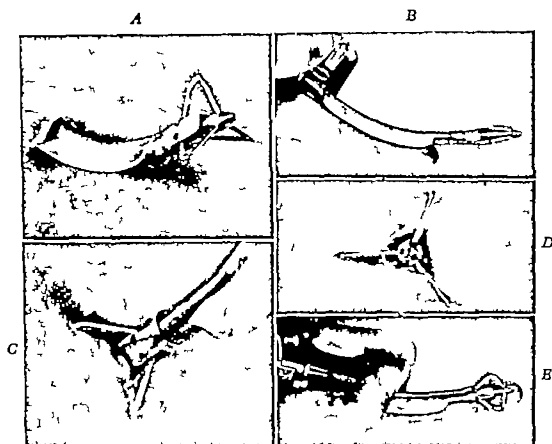


Fig. 1 Dilator for aortic stenosis.

geon s hand and the index finger of the opposite hand lies over the shank, with the tip of the instrument at the point where the commissurotomy is to be accomplished.

By drawing the opened instrument back through the valve, the commissures will be incised and the valve opened (Fig 1*E*) The commissures truly "line up' with the expanded blades, because the index finger tip is seated in a sinus of Valsalva.

Figure 6 shows the acquired rheumatic aortic valve stenosis with secondary calcareous change. Although these commissures appear to be somewhat open, immediately beneath their leading edges one finds little cross strands of calcific adhesion. Upon occasion, as Dr McMillan indicated, the mere touch of a finger, or anything else, to such a fusion will cause it to split open.

However, one of these commissures was very tightly adherent, and the total area of opening, before any commissurotomy was effected, was indeed very small, lending emphasis again to the statement of Dr Bailey, that even though we open only one or two commissures, and not all three, we do get an effective increase in the cardiac output of the patient.

In Fig. 3 the instrument is in place, about to be pulled out to accomplish



Fig 2 Rheumatic aortic valve stenosis.



Fig 3. The di[illegible] n place in the valve

the commissurotomy. This h[illegible] d on 11 autopsy cases of aortic stenosis without the surgeon ac[illegible] the valve, merely feeling it, and in all ca[illegible] mmissures we[illegible] op[illegible]

There are a variety of acting attachments (Fig 4) which are available to the cardiac surgeon, as additions and refinements to the original aortic dilator

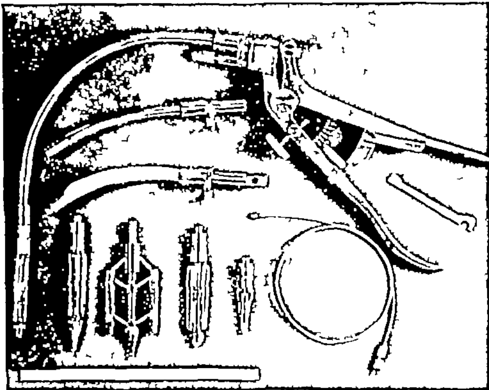


Fig. 4 Attachments for the aortic dilator

William K. Swann (*Knoxville, Tennessee*)

We have previously reported that by using a plastic operative tunnel it is possible to reduce the formidable aortic operation to the simplicity of the mitral operation.

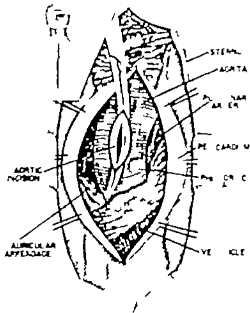


Fig 1 Incision for use of plastic tunnel.

A sternal splitting incision is used (Fig 1). A lip of the aorta is isolated. A bifurcated or single channel tunnel may be used.

Figure 2*A* shows digital exploration of the aortic valve, and section of aortic commissures. This illustrates the single channel tunnel which we now use and which is just as effective.

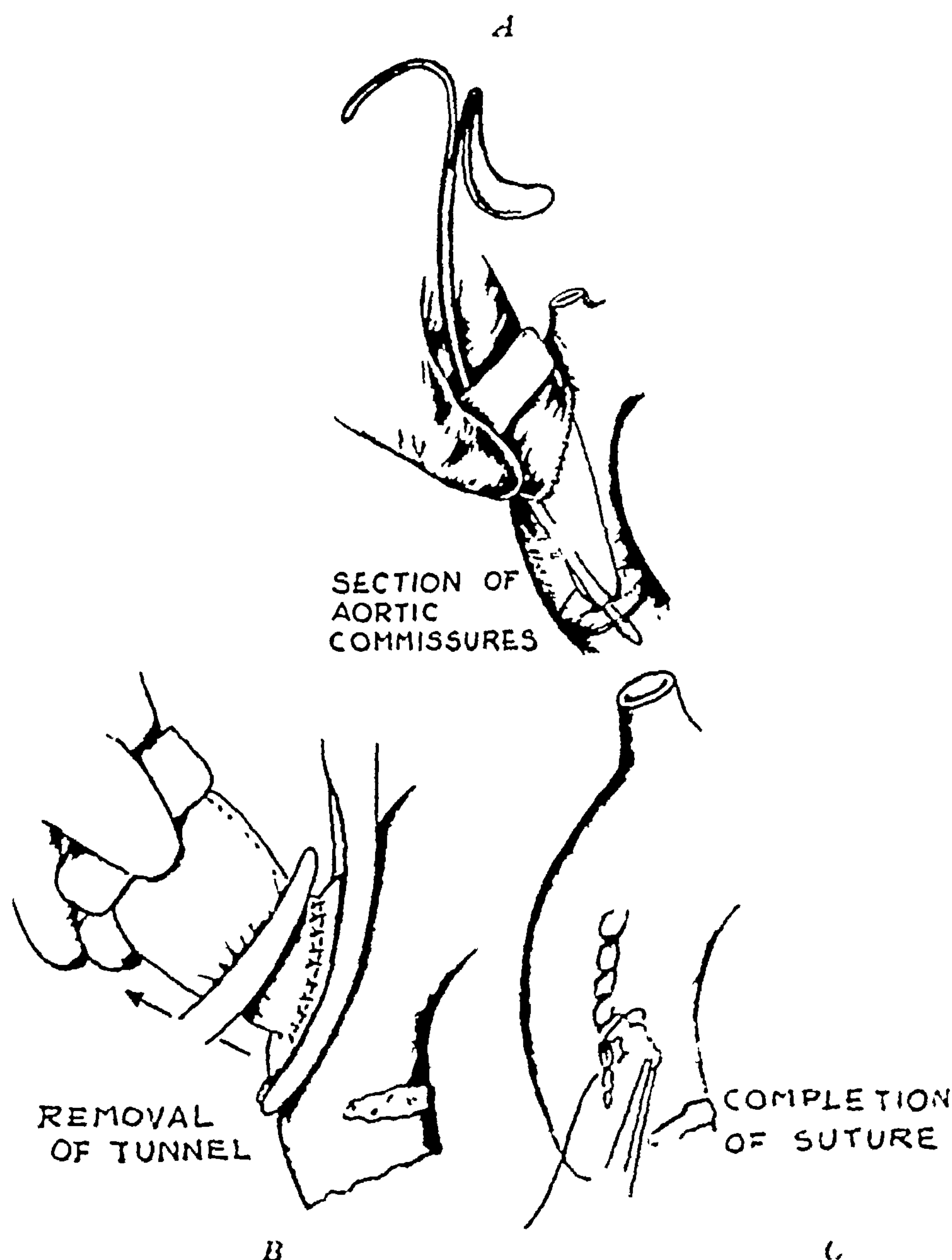


Fig 2 *A*, Digital exploration of the aortic valve and section of commissures *B*, Removal of tunnel *C*, Completion of suture

Figure 3 illustrates removal of the tunnel, and also serves to illustrate how it is sewed to the aorta Figure 4 shows closure of the aorta

We feel that surgeons who are experienced in the mitral operation can perhaps do the aortic operation with not a great deal of difference in immediate mortality and morbidity We would caution against using any instrument from above without accurate finger guidance, inasmuch as the [illegible]

ing may be eccentric and smaller than the opening of the coronary arteries

We also feel that perhaps bacterial endocarditis may be more common after aortic valve surgery than other valve surgery We have seen one case in our small series, and one case operated on elsewhere, and we therefore use large doses of antibiotics prophylactically, and we avoid the use of the ungloved finger

Another precaution is that the clamps on the aorta may result in late aneurysm formation, and perhaps the incision should be reinforced with a sheet of nylon cloth.

(4) A tube placed through the apex lines up neatly with the long axis of the left ventricle and thus provides the most hydraulically desirable outflow tract.[4] (5) This position precludes obstruction of the tube by the septum during systole.

A method has been developed for introducing the prosthesis into the apex of the ventricle which

(a) involves little or no blood loss,
(b) requires 30 to 60 seconds for its completion,
(c) eliminates the hazard of left ventricular and coronary artery air embolus,
(d) assures that the insertion is made precisely into the ventricular apex through the apical dimple and
(e) may be done, if desirable, without the use of any myocardial sutures

Jeger, cited by Kuttner, apparently kept one dog alive for four days following ventricular-aortic by-pass with a venous segment and occlusion of the ascending aorta.[5] Bailey, Glover, O Neill and Redondo-Ramirez redirected their attention to the diseased valve itself after being "discouraged by these attempts at by-passing or replacing the aortic valve."[6] Hufnagel reported that his attempts at ventricular-aortic anastomosis ' have had little success "[7]

PROCEDURE

INSTRUMENTS The valve prosthesis used in these experiments is shown in Fig 1 * The ventricular end has an inside diameter of 11 mm. (cross-

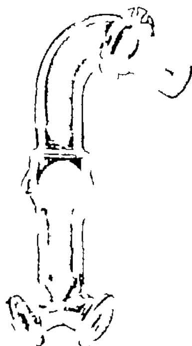


Fig. 1 Valve prosthesis used in the AAA procedure. The ventricular tube end has been shortened to 11 mm. instead of the 16 mm. shown.

* The authors are indebted to Mr Carl Hewson Brunswick Mfg. Co, for the fabrication of the prosthesis

sectional area 0.95 sq. cm) and an outside diameter of 12 mm. The ventricular end of the tube is 11 mm. long and it is this segment which extends from the external surface of the apical myocardium into the lumen of the ventricular cavity. Just distal to this segment there is a small ridge which rests on the epicardium and there is also a freely rotating spoked wheel The latter is used to affix sutures to the myocardium, the pericardium or both.

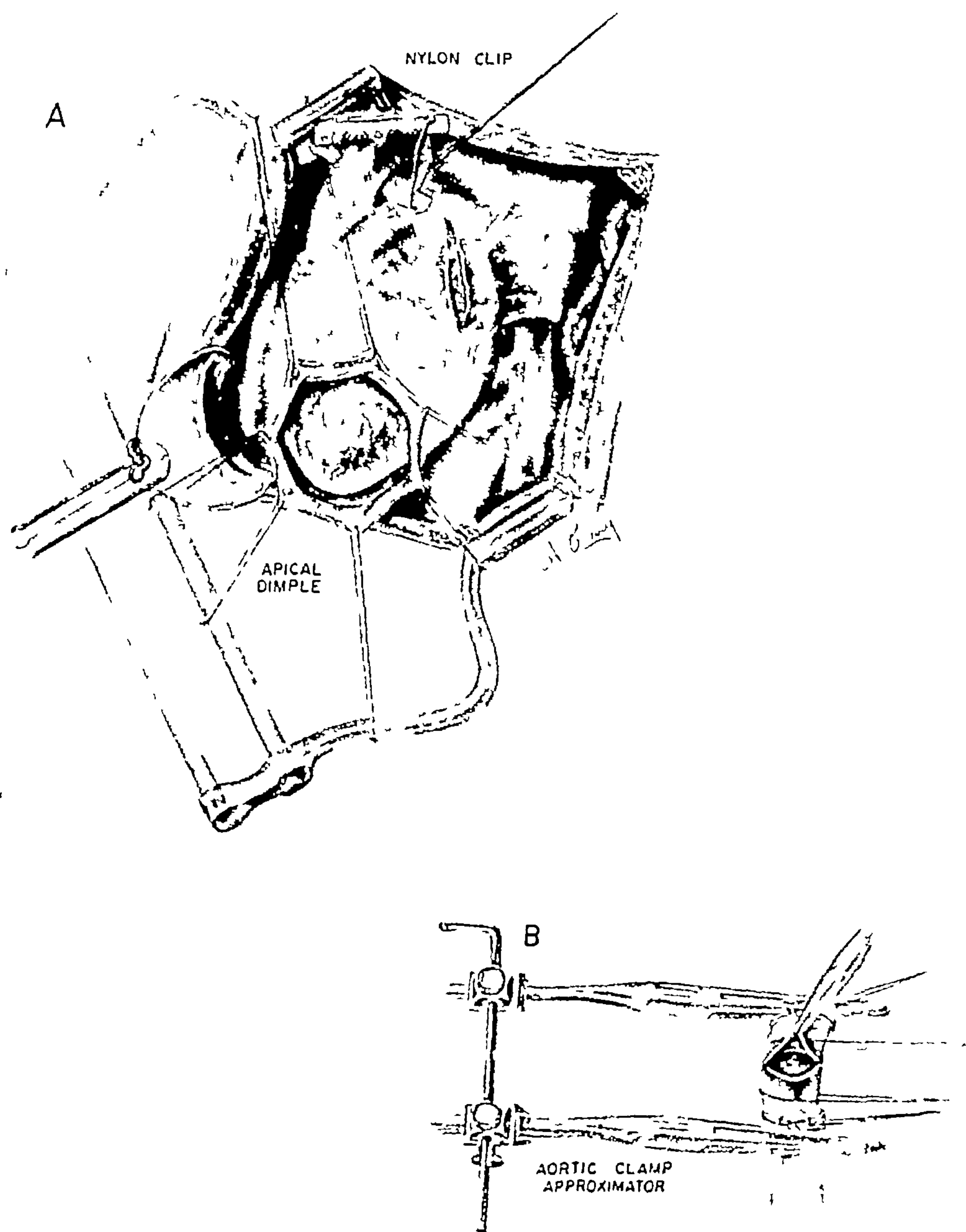


Fig 2 *A*, Aorta is mobilized, the pericardial aperture is formed, the pericardium is slit over the left atrium and the nylon clip is in place on the ascending aortic arch Note the apical dimple, a whitish, dimpled spot which is clearly present in about 3 out of 4 dogs When not clearly present visually, its location can be ascertained by exploratory dimpling with the tip of the fifth finger It signals the precise location of the true left ventricular apex The phrenic nerve has been omitted *B*, Gross aorta clamps on the thoracic aorta are drawn together with the aortic clamp approximator, producing slack in the segment to be intubated with the Y end of the valve prosthesis Multiple point suspension rings and ligatures are in place

After the tube curves and leads the blood through a valve of the Hufnagel type,[8] it leads to the wide-angled Y-shaped aortic end. One end of the Y tube conducts blood cephalad, the other caudad. The internal diameter of each arm of the Y tube is 10 mm., the outside diameter is 12 mm. Two grooves

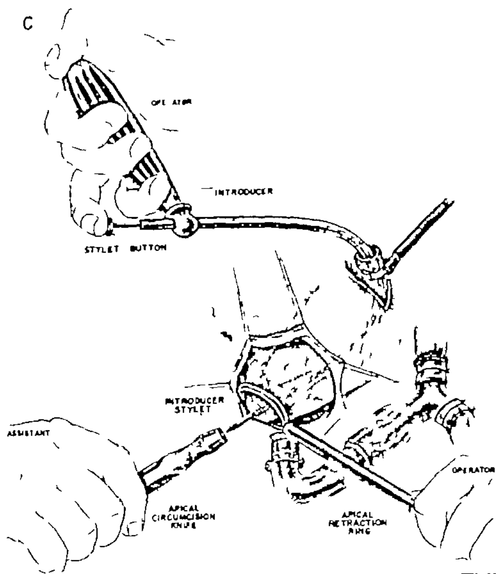


Fig. 2. *C* Valve prosthesis intubation into aorta has been completed and prosthesis filled with saline and stoppered. Introducer has been placed into left ventricle via the left atrium and the apex is drawn over the end of introducer with the apical retraction ring. The introducer stylet has been made to pierce the apical dimple by pressure on the stylet button. Assistant is preparing to slide the apical circumcision knife over the stylet and cut out plug of myocardium.

are present on each arm of the Y tube, a number 2 silk tie fixes the aorta in the proximal groove and a Hufnagel,[8] nylon, multiple point suspension ring fixes it in the distal groove (Fig 2)

The instruments required to perform the apical aortic anastomosis (AAA procedure) as outlined below are shown in Fig 2 The *introducer* (2*C*) is a curved stainless steel tube bent to the shape shown with a handle on it. The

apical end of the introducer has on it a Lucite bulb the end of which in turn is capped with a rubber piece fixed onto it with a silk ligature A stylet runs the length of the tube and terminates in a long, sharp, trocar point which emerges 35 mm. from the apical end of the introducer when pressure is made on the stylet button

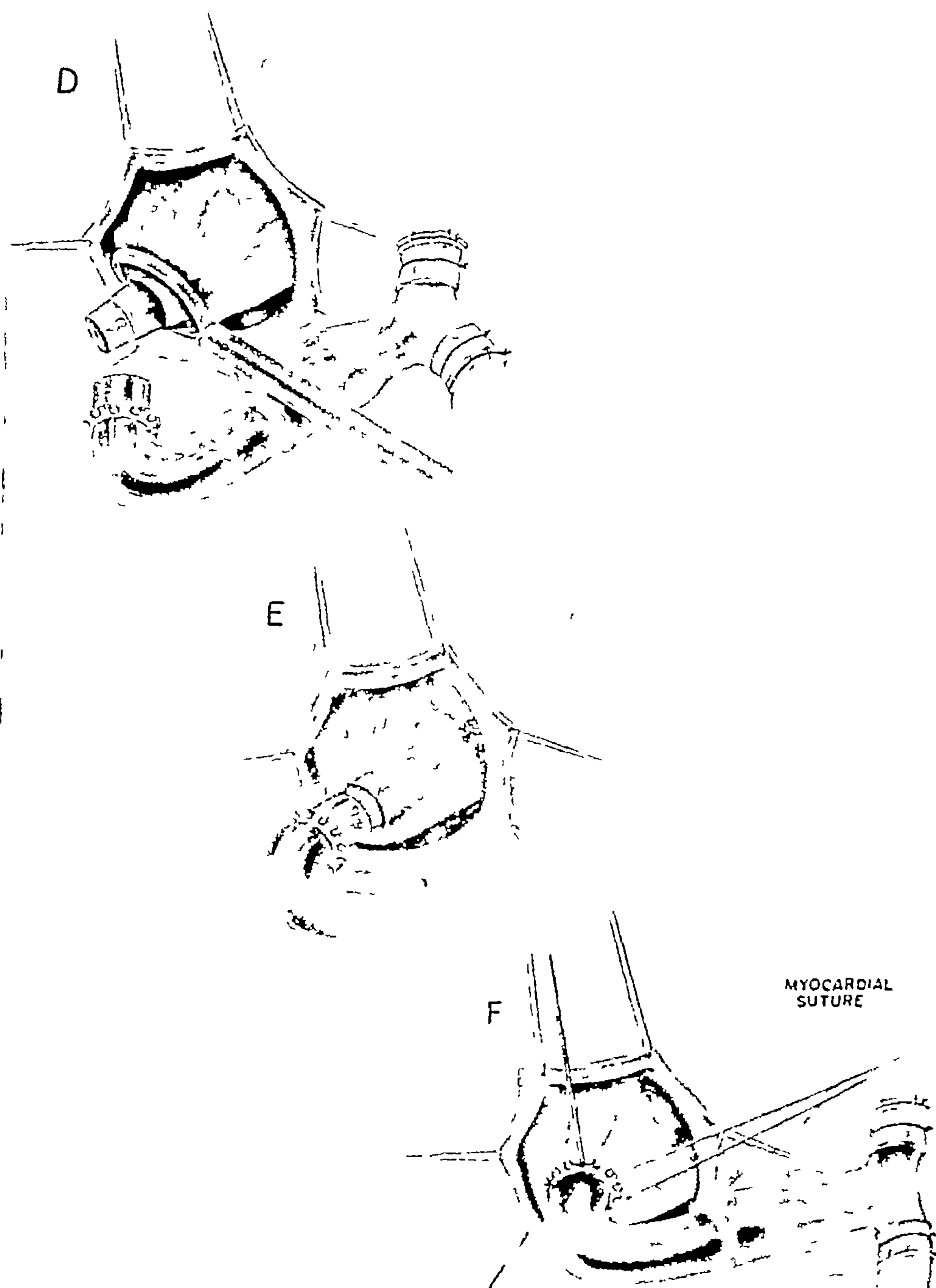


Fig 2. *D*, Myocardial plug has been cut and removed and stylet has been retracted Stopper has been removed from the prosthesis and the coupling of it to the introducer begun Not shown is the catheter which plays a stream of saline into the prosthesis as the coupling proceeds *E*, The prosthesis has been coupled onto the introducer and is being drawn into the left ventricle *F*, Ventricular intubation is completed. Myocardial sutures are being affixed to the spoked wheel

The *apical retraction ring* (2*C*) is used to retract the apex of the left ventricle back over the end of the introducer as the latter seeks the internal apex. This simple ring serves three highly important functions. First, it prevents the dislocation and overstretching of the heart and ventricle, thus abolishes or greatly diminishes the arrhythmias which are observed if it is not used. Second, it stretches an isolated segment of the apex over the end of the introducer, steadies it, and delineates the precise spot (apical dimple) where the myocardial hole is to be cut. Last, it enables the introducer to be gently and precisely persuaded through the *small hole cut in the apex by coning the* apex down over the tip of the introducer.

The *apical circumcision knife* is an 8 mm. dermatology biopsy punch renamed for this purpose. The only way in which it has been altered is the provision of a hole up its middle long enough to accept the full length of the pointed stylet after the latter has emerged from the end of the introducer and has pierced the apex (2*C*).

The *aortic clamp approximator* is shown in Fig. 2*B*. Its function is simply to draw together and maintain parallel the two Gross aorta clamps after the latter have occluded the thoracic aorta, thus providing a slack rather than a taut aorta during the intubation of that vessel.

STEPS IN THE PROCEDURE. 1. Under Nembutal anesthesia and intermittent positive pressure breathing, the 6th rib is removed* with the dog in the right lateral position. The artificial respiration device (Starling pump) is so adjusted by means of a screw clamp resistance on the expiratory line that *there is* an end-expiratory pressure of *at least 4 cm. H_2O*. Permitting the lungs to collapse further than this during expiration produces arterial hypoxia.

2. The right and left intercostal arteries, 1 through 4 or 5, are ligated, divided, and the aorta mobilized.

3. The nylon clip, shown in Fig. 2*A*, is then put in place across the ascending arch of the aorta 2 to 3 cm. above the aortic valve just proximal to the brachiocephalic artery. This clip is to be permanently closed immediately after the AAA procedure is completed, thus insuring that the cardiac output (minus coronary flow) will leave the ventricle via the apical route.

4. A pericardial aperture is formed by incising the pericardium near the apex and retracting it with sutures tied to the Balfour retractor as shown in Fig. 2*A*.

5. A second pericardial incision is made lateral to the left phrenic nerve over the site of the left atrium. The atrial appendage is then picked up, and the noose of a Rumel tourniquet slipped over it and slid well down over the atrium, care being taken not to impinge on the left *circumflex coronary artery*. The appendage is opened, picked up in four fine hemostats and the trabeculae cleared so as to permit the ready insertion of the introducer.

The foregoing steps are completed in an unhurried fashion. The subsequent sequence, *however, should* be *performed* expeditiously so as to (*a*) keep the time of total thoracic aortic occlusion under 10 minutes, and (*b*) limit the time of the ventricular intervention.

* *More recently an incision in the 5th interspace has been found to be adequate*

6. Two Gross aorta clamps are placed 8 to 10 cm. apart on the thoracic aorta and closed. They are then brought closer together and fastened in place with the aortic clamp approximator so as to give the aorta some slack. The aorta is then transected between the clamps and the aortic intubation is completed. A No. 2 silk ligature ties the aorta securely in each proximal groove and a multiple point suspension ring fixes the aorta in each distal groove (Fig. 2*C*). The prosthesis is then filled with saline through a catheter inserted in the ventricular end. When the air has been exhausted therefrom, a soft rubber stopper is placed in the ventricular end of the prosthesis and the aortic clamps removed, thus permitting resumption of aortic flow. The time of thoracic aortic occlusion varies from 5.4 to 8.4 minutes with a mean of 6.9 minutes. This is comfortably under the critical limit for avoiding postoperative spinal cord damage.[8]

7. The introducer is then promptly inserted into the left ventricle via the atrium, seeks the apex and has the latter drawn down over it with the apical retraction ring (Fig. 2*C*) Pressure is then made on the stylet button causing the stylet point to pierce the apex. While the apex is thus steadied by the introducer on the inside and the retraction ring on the outside, the apical circumcision knife is slid down onto the protruding stylet (Fig 2*C*) and cuts out a circular plug of apex. The plug thus cut out is removed, the stylet is retracted and the introducer eased out through the apical hole with the help of gentle counter-pressure from the apical retraction ring (Fig. 2*D*).

8. The stopper previously placed in the ventricular tube end of the prosthesis is now removed and the tube coupled onto the soft rubber end of the introducer. The function of the soft rubber tip is to preclude the possibility of scratching the internal surface of the prosthesis (Fig. 2*D*, *E*). Air is exhausted from the tube by a stream of saline from a catheter as the coupling is completed

9. The tube is then drawn back through the apical hole with the introducer (Fig. 2*E*), and after the tube is well in the ventricle the introducer is disengaged from the prosthesis and gently withdrawn from the heart.

As more confidence and facility with these maneuvers was acquired, without any particular sense of urgency, it was possible to complete steps 7, 8 and 9 (Figs. 2*C*, *D*, *E*) in 24 to 65 seconds with a mean time of 46 seconds.

10. The nylon clip previously placed around the ascending arch is closed with a No 2 silk ligature

11. The atrial appendage is doubly ligated

12. Four or five 00 sutures are then placed in the myocardium immediately adjacent to the tube and fixed to the spoked wheel (Figs. 1 and 2*F*). The edges of the pericardial aperture are then affixed firmly to the spoked wheel by means of additional sutures. In two of the surviving dogs the myocardial sutures were omitted and the tube held in place solely by pericardial sutures.

The end result of this procedure is a 12 mm. Lucite tube in an apical hole produced by an 8 mm. circular knife. The fit is sufficiently snug to prevent leakage around the tube. The removed myocardial plug measured 5 to 6 mm in diameter and weighed an average of 170 mg. or approximately 0.1 per cent of the estimated total heart weight.

The dogs weighed from 18 1 to 36 8 kilograms

Figure 3 is a roentgenogram taken three months after operation and shows the position of the ventricular end of the prosthesis The ascending aorta is occluded. Figure 4 shows the femoral arterial pressures registered in 4 dogs after completion of the AAA and occlusion of the ascending aorta. Figure 5 shows the smoothly lined apical outflow orifice after removal of the prosthesis 2.2 months after operation.



Fig. 3 Roentgenogram taken three months after operation showing ventricular end of prosthesis in the left ventricular apex. Ascending aorta occluded.

Eight dogs have survived the above described procedure for from three to ten months and appear to be in vigorous good health. The operation itself is technically not difficult and can be consistently accomplished in the dog Two types of postoperative complications have, however, produced an over-all mortality which precludes the present clinical application of the AAA procedure in the form described above.

The first of these is the intravascular hemolysis and the red cell destruction produced by the Hufnagel valve in the AAA position in the dog This is evidenced by hemoglobinemia, hemoglobinuria and the heavy renal deposition of hemosiderin. Cr^{51} tagged red cell observations reveal that red cell survival is substantially shortened. This matter is treated in greater detail elsewhere;[9 10 11] the steps being taken to diminish or eliminate this hazard will be described below

of these studies two of the dogs who had had the AAA procedure were brought home as house pets At first one or both were permitted to sleep at the foot of the bed. It became clear that even if the patient does not mind it, the spouse might.

The design of a ball valve with a slightly elastic housing was therefore undertaken in the hope that the impact of the ball would be diminished by the resiliency of the valve housing and thus make the valve both quieter and

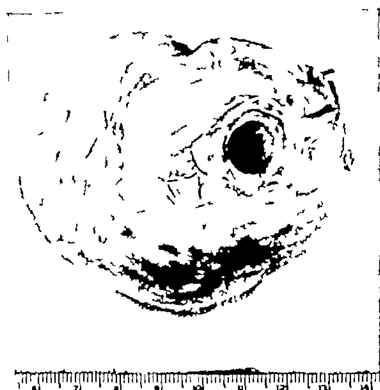


Fig. 5 Apical orifice 66 days after operation. Pericardium has been cut away and prosthesis removed.

less traumatic to the red cell. The result is shown in Fig 6 The ball is of Lucite but the entire valve housing is of elastic silicone. The composition of the elastic silicone is such that, when made with a quite smooth inner surface, it is refractory to clot. At the pressure gradients which occur in vivo it is less regurgitant than the Hufnagel valve and is substantially quieter It is appreciably less costly to fabricate. Perhaps most important of all, it produces only a small fraction of the red cell destruction produced by the Lucite ball in the Lucite housing An elastic silicone ball may also be used. Simple elastic silicone tubes have also been placed in the thoracic aorta in 6 dogs They appear promising as a blood vessel substitute.

In one sense the title of this communication partially belies its fundamental intent. Rather than an attempt to alleviate aortic stenosis per se, its purpose is to examine a different approach to the broad problem of alleviating cardiac valvular disease, to examine its hazards and to speculate about its possible merits Valve replacement has several possible advantages. First, it provides in advance a precisely known outflow resistance. Second, the provision of a

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DISCUSSION

Dwight E. Harken (*Boston*)

We can recognize the terminal phase of aortic stenosis This should not forever remain the indication for surgery It is interesting that Dr Laurence B Ellis traced 100 patients from autopsy backward through their clinical course and substantiated many of the things you already know He also brought these things into sharper and more dramatic focus We all know that left ventricular failure is a very grave sign. However, he found that atrial fibrillation is similarly significant. When fibrillation or failure was followed by heart pain or syncope, the patients were dead within weeks or months The reverse does not hold. Patients occasionally go for a long time with syncope or angina. However, patients die promptly when failure and/or atrial fibrillation are compounded by pain or syncope.

Dr Ellis findings correspond with our personal experience in 34 patients

in the last four years, for whom I recommended aortic surgery because they were developing left ventricular failure, syncope or angina yet who did not accept operation. Of those 34 unoperated patients, 30 were dead in less than six months. So much for the horrible prognosis in this disease.

There is no question in my mind that the surgical approach to aortic stenosis is from above through the aorta. In this way we can do quite well by a large majority of these patients.

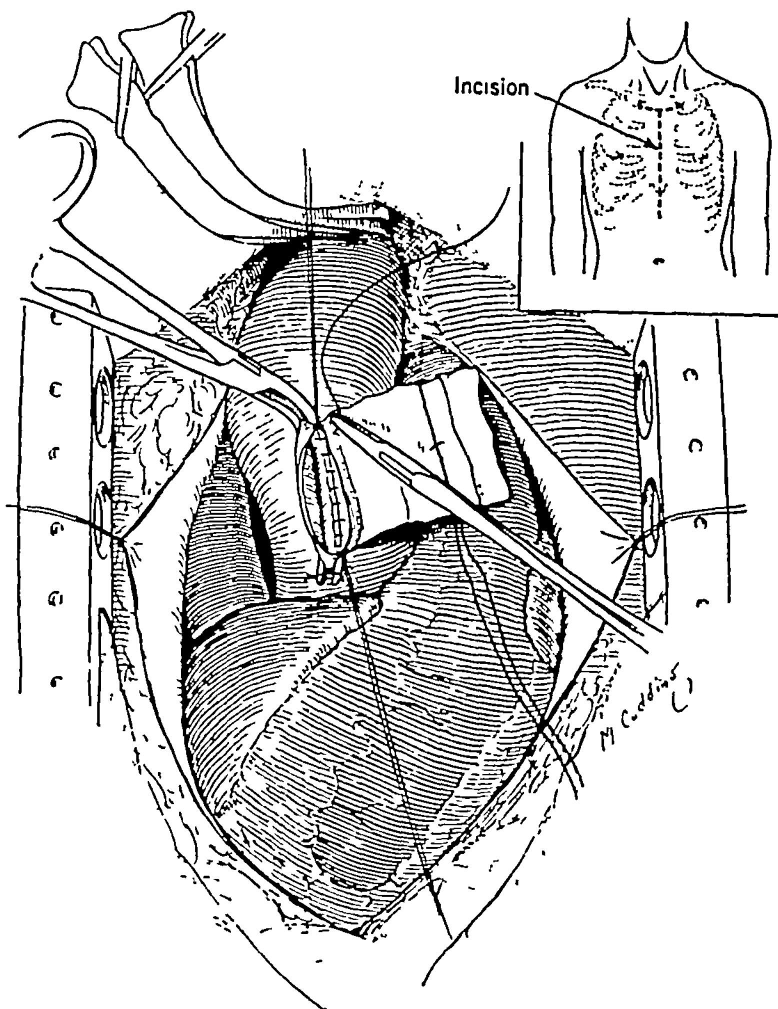


Fig 1 Swann pouch used as operating tunnel.

We use Dr Swann's approach. A knuckle of aorta is raised on an Atraugrip clamp. The side-tracked zone of aorta is opened and a pouch is sutured to this In the illustrations you see Dr. Swann's pouch with a shoe-string sewn to its base This constitutes a good operating tunnel (Fig. 1).

Over the years we have collected a color film library of fresh specimens from patients who have died from aortic stenosis. In color moving pictures, one preserves color and texture. Furthermore, by hooking a long series of such film strips together and running through them at one time, one sees patterns that he would otherwise not observe. Here are the general types we have seen (Fig 2).

Type A, or the functionally bicuspid form, is present in approximately 60 per cent of the cases. Type B was encountered in more than 20 per cent.

Type C "text-book' form was seen in less than 15 per cent. Formerly in Type A, the calcific bicuspid bars were broken in the middle whether there were previous commissures or not. However, such clumsy technique was associated with regurgitation and fragmentation with embolus It is preferable when possible to fracture laterally to the annulus Having effected this fracture we try to extend this lateral fracture both fore and aft to mobilize the bicuspid stenotic bars as a trap-door When this is not possible by direct finger fracture, it is frequently possible to use one or another of a variety of valvulotomes I happen to use our little No 9 bread-knife valvulotome that we ordinarily use for mitral stenosis

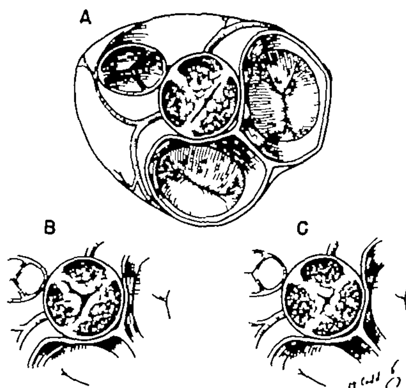


Fig. 2. Varieties of patterns of aortic stenosis.

The form of aortic stenosis, so much discussed in this volume, Type B, has been observed in patients who died from aortic stenosis in something over 15 per cent and the Type C in about 10 per cent. Type B is a highly favorable variety for finger fracture and we have corrected it by the trans-aortic approach almost perfectly Type C is least favorable but practice improves the results just as it improves the efficacy of a stone-cutter's fission.

There is substantial reason to believe that until Dr Sarnoff is able to establish his ingenious procedure, there is some room for the direct attack in aortic stenosis along these lines

To any of you who have used both approaches it will seem as unreasonable to continue the transventricular approach as it is to perform mitral valvuloplasty for mitral stenosis blindly through the ventricle as was done many years ago

E. H. Fell (*Chicago*)

Aortic stenosis is a treacherous disease for which medical therapy has very little to offer. It is evident from the paper just presented and the discussions concerning this subject that surgery for aortic stenosis, at the present, is far from ideal For the past two years we have dilated stenosed aortic valves by a procedure that likewise is not ideal, but the method has some virtues that are, we believe, worthy of consideration. Because of the high incidence of difficulties associated with dilation of the aortic valve by way of the left ventricular route we elected to approach the valve by way of the aorta. Mr. Bruno Richter of Lombard, Illinois, created a small strong instrument, the tip end of which is 5 x 2 mm. Dilation is possible up to 25 mm. by means of a blunt two-plane dilator (Fig 1).

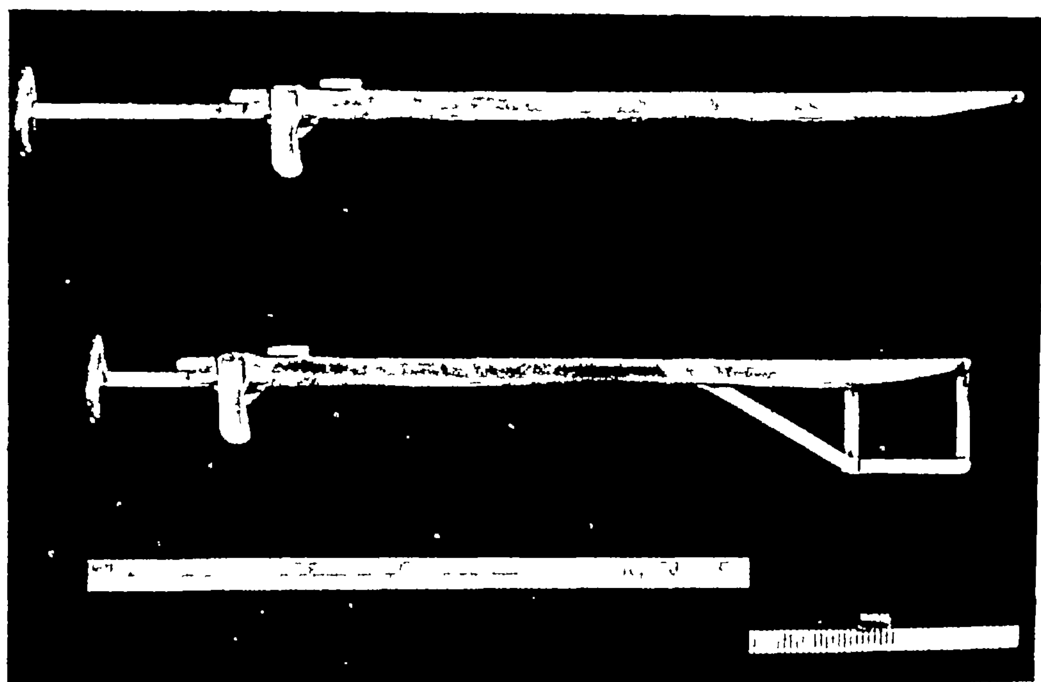


Fig 1 Photograph of instrument used in dilating the aortic valve by way of the retrograde route.

The aorta is well exposed by splitting the sternum in its entire length. The first portion of the aorta is freed from its mediastinal attachments from its origin to the innominate artery A site on the ascending aorta is chosen (Fig 2) for insertion of the dilating instrument, approximately 3 to 4 inches distal to the valve, in line with the valve Two approximating stay sutures of silk are placed at this site, and in between the sutures a small incision is made in the aorta to allow for the insertion of the instrument After insertion the approximating sutures are drawn taut by an assistant, thus very little blood occurs at this site The surgeon now places his thumb and index finger of the left hand at the level of the aortic valve and thus aids in directing the instrument gently through the stenosed opening into the left ventricle. There should be no forcing or pushing of the dilator.

In the 10 cases dilated, the dilator first contacted a hard firm valve, but with a little change of position, and particularly at systole, the dilator drops

through into the ventricle without difficulty The dilator is then opened and brought back against the stenosed valve. The left index finger and left thumb can feel the valve open (Fig 2) as dilation takes place. A number of dilations may be necessary The instrument may be rotated if desired, however, the *line of least resistance* will give when a strong blunt dilator is used. A Satinsky clamp is placed on the aorta at the site of incision as the dilator is withdrawn. The incision in the aorta is closed.

One calcified stenosed valve could not be dilated by this method, nor could it be benefited by direct finger fracture. In 10 others, from 2 months to 55

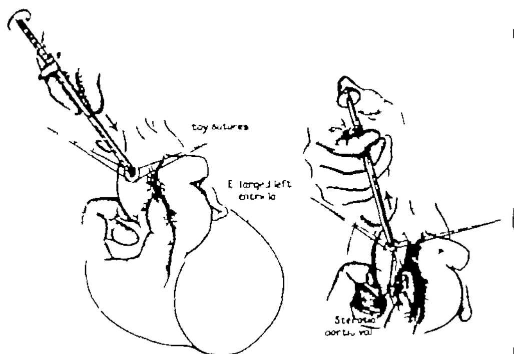


Fig 2. The sketches illustrate the technique of dilating the aortic valve.

years of age, this method of dilation was possible. Two patients died on the operating table. The first death occurred following attempted dilation by instrument and finger and the dilation was impossible, as previously stated. The other patient, 50 years of age, had a severe stenosis and died while the chest was being opened.

This method of dilating the aortic valve is not ideal but has these advantages (*1*) the diseased left ventricle is not entered, thus preventing the associated complications of that route, (2) the small strong instrument used can be effective in dilating the valve yet produces little trauma to the aorta, (3) with the fingers of the right hand gently guiding the instrument and with the index finger and thumb of the left hand at the level of the aortic valve, dilation of a stenosed valve can be accomplished in most all cases

CONCLUDING REMARKS

Charles P Bailey (*Philadelphia*)

It has been suggested that the mitral valve operation in combined mitral

and aortic stenosis from the right is handicapping. Actually, we feel we can do a better technical procedure from the right than from the left. The posterior commissure is much easier to attack from the right than from the left, and in the last 60 cases of isolated mitral stenosis we have operated on, we have used the right approach electively because we not only can treat a previously unsuspected tricuspid stenosis (which is present in 5 per cent of these cases), but also the aortic stenosis, if it is present.

One could also fix an interatrial defect. Above all, one avoids the clot in the left auricular appendage, which was present in 25 per cent of our entire 1500 operations for mitral stenosis. We think that is a better approach, because we can take care of all three valves at the same time.

Dr Muller emphasized the value of earlier operation. That is something we will have to get our medical confreres to accept. It is certainly the right philosophy of treatment.

Is there a possibility of subacute bacterial infection with bare-handed technique? Statistically it is no more common than when we used the glove in our previous series We have used it for eighteen months for all intracardiac surgery. However, there is a greater incidence of subacute bacterial infection when you operate on the aortic valve than any other valve, and that may be due to the trauma and high pressures concerned.

I think Dr Sarnoff is a little cruel when he says we produce stenosis when we relieve aortic insufficiency by tightening the annulus or by putting in a stent We do to a certain extent, but we get a net gain. If he can do the operation with no mortality, since it renders the patient immune to the effects of rheumatic fever on the aortic valve, maybe we ought to have it done more or less prophylactically if we have had any evidence of rheumatic fever.

SURGICAL TREATMENT OF AORTIC INSUFFICIENCY

CHARLES A. HUFNAGEL (*Washington, D C*)

The problem of aortic insufficiency seemed a very formidable one when we first began our investigations of this problem some seven or eight years ago At that time our knowledge of the entire problem was at great variance with what we had learned in the subsequent years

Initially we felt that one of the good approaches to this matter might be homologous transplantation of another valve; and since we had been working so closely in relation to transplantation of arteries at that time, we made many attempts to do this

The initial response of such a homotransplant of the aortic valve is ordinarily excellent. There are certain technical problems about it in that the base of the aortic valve actually lies in the muscle of the ventricle rather than in the base of aortic tissue, per se, but these are not difficult to overcome. Unfortunately, long-term results of such transplantation have not been as satisfactory as we would have liked. Grafts of vein, pericardium and so on, and valves constructed from pieces of aorta or other vessels, have gradually shown the effects of wear and tear over the long run, and so we have gradually abandoned them.

It then seemed that our best possible solution to the problem might be the development of some inert valve, and so the present work was undertaken.

Before discussion of our procedures, I think it might be worth while to point out that the natural history of aortic insufficiency has not really been well known, or has not been well understood. One should understand, too, that aortic insufficiency in which there is a high systolic pressure, but with good maintenance of a high diastolic pressure, is not the same kind of situation as that in which there is some elevation or perhaps even major elevation of the systolic pressure with a diastolic pressure under 40 mm. of water by direct measurement. One should not be confused by this type of situation in the evaluation of patients

This discussion is actually confined to patients who have free, wide-open aortic insufficiency The etiology in the patients we have treated has been rheumatic, luetic, traumatic, congenital, and acute or subacute bacterial endocarditis with rupture of a cusp

It also has not been well recognized that patients who have free aortic insufficiency are like *those with aortic stenosis, that is, liable* to sudden death. Patients who may be apparently relatively well compensated on a good med-

ical program may die quite suddenly of ventricular fibrillation. This relationship to coronary flow and to cardiac work is now becoming more and more apparent.

Of the patients whom we have had an opportunity to study, some of whom have been operated on, and others either followed medically or backward from the post-mortem room, and in checking their clinical histories and physical findings against their post-mortem findings, it has been rather surprising to us to find how uncommon is the association of major degrees of aortic stenosis in the patient who has wide-open aortic insufficiency. It also has been rather interesting to observe that in this same group of free aortic insufficiencies real mitral stenosis of a serious or significant degree is also relatively uncommon.

It is very often difficult to differentiate clinically whether or not true mitral stenosis is present. Earlier in this work we often explored patients in whom there was doubt, and to our chagrin we found that very often the diagnosis of mitral stenosis was incorrect, and the murmur that had been heard was actually the murmur of aortic insufficiency.

In this general group of patients with whom we have had experience, I will speak only about the first 80 cases that have now gone sufficiently long so that something can be generally told of their over-all situation. In this group 50 per cent were terminal patients, that is, patients who had severe failure and failure which had been extremely difficult or impossible to control medically. The over-all prognosis in this group of patients is, of course, very bad without surgical intervention.

No patient has been accepted for operation who has not had some signs of decompensation. All of the patients have had wide-open aortic insufficiency. None has been rejected because of concomitant lesions No patient has been rejected because of too severe disease, providing he could survive the trip to the operating room.

It has been interesting, too, to see that almost all of these patients have had signs of angina pectoris, and in some of these status anginosus has been present In a great number there has been a loud systolic murmur at the apex which has been suggestive of mitral insufficiency.

Again by exploration and by other autopsy studies on patients who have exhibited a murmur in life, it has been found that an organic deformity of the mitral valve has been a relatively uncommon situation, and when it did occur it has usually been relative mitral insufficiency associated with dilatation of the mitral ring with left ventricular dilatation.

We will proceed now to the discussion of the type of valve which has been employed in these studies.

Figure 1 shows a valve which has its chamber made of a single piece composed of methyl methacrylate. The fixation rings at the ends of the picture are made of nylon. The teeth, which project downward, are made in such a fashion that the length of the teeth is greater than the thickness of the aortic wall. This is of extreme importance. The little holes which you see are for the insertion of an instrument which will close the ring around the aorta and will hold the valve in place as soon as the instrument is closed.

split in the ring is placed there so that it will be easy to place the ring
d the intact aorta before it is sectioned.
e flow factors have been so calculated in the valve that the available
e is approximately twice the area of the inlet, so that the resistance
s, and the like, created by turbulence in the system because of the
nce of the ball, are minimized, and the total over-all resistance is
nal.

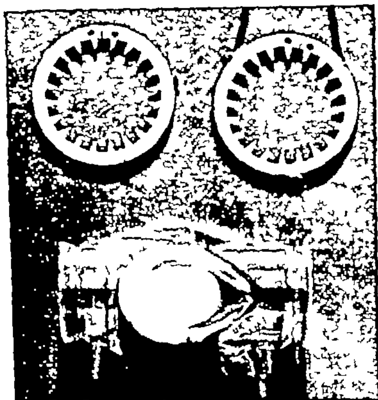


Fig. 1 Aortic valvular prosthesis with its multiple point fixation rings

arly in the work, when we tried to convert from the dog's size valve
h which we had worked for several years) to the human size, we were not
ing this chamber adequate and we were getting some evidence of ob-
ction. This, of course, can be overcome by increasing the size of the
pass passages
he shape of the ends is important, as is the polish. Currently and for the
year and a half we have made the ball also out of methyl methacrylate
ause we felt this surface is better than the polyethylene ball shown here.
ote the area of fixation. The tip of the lip is in contact with the aorta.
s has been removed approximately twelve days after its insertion. There
elatively little reaction to the end of the valve. However, if this valve is
essively sharp, or if it is not properly placed, a traumatic arteritis can be
up by the motion of the valve in the aorta.
Ve call this principle of using the fixation rings "multiple point fixation,"
the principle involved is primarily the use of multiple points and the
idance of complete circumferential pressure. This allows the blood supply
he distal portion of the aorta to be preserved so that this does not become
roblem.

However, if one does not have a proper fit and the valve is actually too small for the size of the aorta into which it is inserted, the aorta may bulge and come into contact with the ring. In this instance one will almost certainly get erosion of the aorta and aneurysm formation So, one must be careful that he does not accept merely the principle without understanding its real application.

More recently we have been placing a small band of loose, elastic Orlon around the ends of the aorta outside of the aorta to make this portion of the aorta hug the portion of the valve inside, which projects beyond the fixation line shown here.

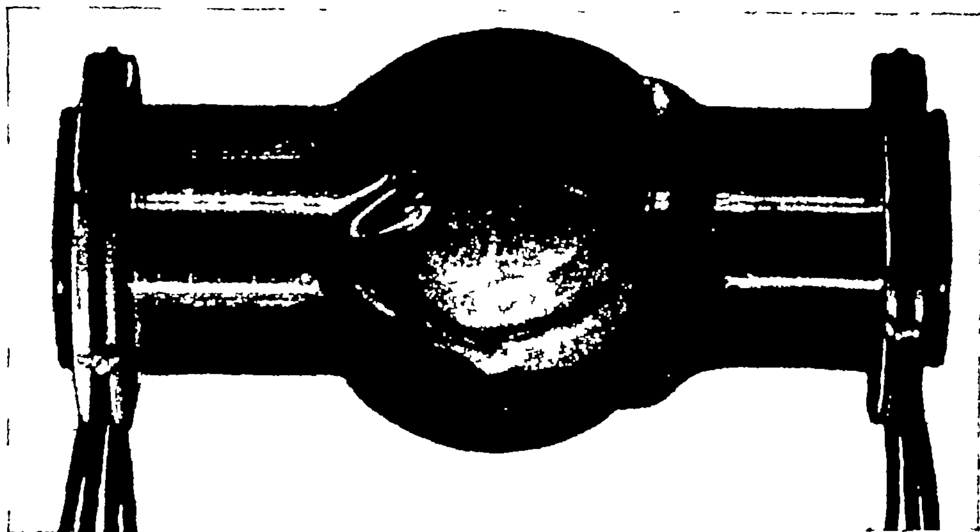


Fig 2 Aortic valvular prosthesis for use in coarctation of the aorta with aortic insufficiency

Early in our experience we had a considerable problem with peripheral embolization. Fortunately, in only one of these instances was this a fatal complication, but we did have other definite and troublesome evidences of emboli. Since we have adopted two measures—the excision of a considerable piece of the aorta so that there is no angulation of the prosthesis, and the use of the Orlon proximally and distally—we have had no embolization. We trust that these improvements will be a major factor in elimination of this problem. We have now gone for approximately six months without embolic complications.

Figure 2 shows a special valve for one of the situations which has been encountered. This is a combination of coarctation (probably with a bicuspid aortic valve) and severe aortic insufficiency. An autograft was used here. The distal portion of the aorta was excised and moved proximally so one could get the valve in in a straight position without angulation and without impingement upon the left subclavian artery.

The use of the valve in this position has certain obvious disadvantages which we fully appreciate, and we have used it only because it has permitted us to operate on patients in whom other procedures have not seemed feasible.

The technique of the operation is shown in Fig. 3. Here you see actually a rather dilated aorta. The vagus nerve is shown here, and the aorta has been

reed from its bed. The dissection is being carried out further with ligation of the intercostal arteries as necessary Ordinarily three or four pairs of intercostals must be divided. The aorta is completely freed from its surrounding tructures The fixation rings are placed before the division of the aorta.



Fig 3 Aorta freed from its attachments for insertion of aortic valve.

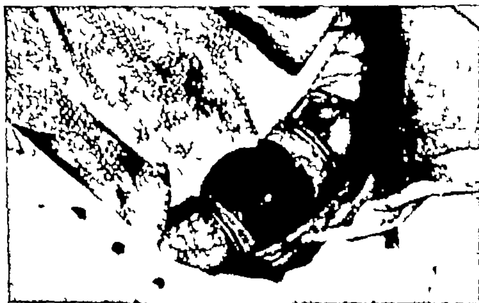


Fig. 4. Aortic valve in place and functioning.

The aortic valve is always available in various sizes These range from 6/8 inch to 1 1/8 inch in diameter for the ordinary type of aorta. We have used all sizes and have a special type of valve for use in coarctation, in which the ends are longer so that one can bridge a bigger gap We have larger sizes available to meet other situations.

Figure 4 is the view immediately after the placement of the valve. Following this, under ordinary circumstances this Orlon sleeve is passed around the

proximal aorta to be sure the bulge of the aorta does not impinge upon the aortic ring and that it does not also move the aorta out from contact with the proximal end of the valve.

We have had one patient in whom an aneurysm developed because of improper placement of the valve. In the other approximately 100 patients no such accident has occurred. We have not in any case failed to be able to place the valve in the aorta.

Figure 5 shows pre- and postoperative roentgenograms taken approximately one month after operation, showing some decrease in heart size. Heart



Fig 5 Roentgenograms before operation and six weeks after operation

size is apparently related to some dilatation of the heart. On some occasions one can actually see the heart size shrink during operation, and a pericardium that has been tight and tense becomes flat and loose after insertion of the valve during the operative procedure.

Management of these very seriously ill patients is extremely difficult. In this group of 80 patients, approximately 12 have not been able to be operated on during the first attempt. They were taken to the operating room, and with the induction of anesthesia the blood pressure fell to such a degree and there were electrocardiographic changes of such a character that operation was deferred. In a fairly large percentage of this group it was necessary to take the patient to the operating room three times before we were actually successful with the use of drugs to sustain the pressure at a reasonable level.

The over-all hospital mortality in this group has been approximately 20 per cent So, actually, in a group of 50 per cent of the patients essentially in the terminal state, there has been an operative mortality of 20 per cent. It is interesting that, through careful management, the actual operative mortality has been relatively small, and that only 5 such patients have been lost during the operation itself.

In most of the deaths the mechanisms were the same, ventricular fibrillation occurring in essentially all instances Fibrillation has begun just about as

often preceding the insertion of the valve as it has following insertion. We have not been able to establish any distinct impression as to whether this occurs following hemodynamic changes from operation, per se, or operation plus insertion of the valve.

While we are continuing to work with other types of valves, we are certainly of the opinion that this has been a valuable procedure, and that individuals who have acute rupture of the aortic cusp from acute bacterial endocarditis, for example, should have immediate operative intervention, since their over-all outlook is so poor



Question How important are embolic phenomena?
Answer Originally they were a serious problem to us, in that we were having emboli in about 15 per cent of the patients In the last six months we have had no emboli.

Question With loss of legs?
Answer No, they have lost no extremities We have had one death.

Question Dr Hufnagel, will you tell us about the noise?
Answer The noise doesn t bother them. The patient becomes so accustomed to it within the first few days that it is no longer noticeable to him except when he makes a conscious effort to listen for it.

The problem in relation to other people hearing the valve is again of no real importance. As long as the patient does not stand with his mouth open and allow the sound to be conducted up the main stem bronchus by air conduction, he has no problem.

PHYSIOLOGIC CONSIDERATIONS RELATING TO THE HUFNAGEL OPERATION WITH SPECIAL REFERENCE TO POSTOPERATIVE ANEMIA

STANLEY J. SARNOFF AND ROBERT B. CASE (*Bethesda, Maryland*)

In considering the significance of certain of the hemodynamic alterations following the Hufnagel operation it is helpful to use as a basis the fundamental relationship between the oxygen requirements of the myocardium and the work it is called upon to perform. In the last analysis it is this relationship which lends meaning to the altered pressures, flows and ventricular work loads that are produced by the operation. Taken together with the penetrating analysis of McKusick et al.,[1] it also provides a rational basis for understanding why the operation is successful in some instances and less so in others.

By means of direct flow-metering and other procedures described in detail elsewhere,[2,3] it was possible to obtain myocardial oxygen consumptions in the same dog over a wide range of ventricular work loads and with different hematocrits and thus determine the relationship of work to oxygen consumption. Data of this type are set forth in Fig. 1.* It is clear that, other things being equal, as the ventricle works harder it requires more oxygen. It would be strange if it were otherwise.

It is desirable to consider one further mechanism, and that is the nature of the control by means of which the myocardium acquires an increased oxygen supply when its work is increased. Since increased oxygen extraction is limited by the already low coronary venous oxygen content, an increase in oxygen available to the myocardium is accomplished largely (sometimes solely) by an increase in coronary flow under normal circumstances.[3] At any given coronary perfusion pressure this is accomplished by a decrease in coronary vascular resistance, that is by vasodilatation.[3] It must also be remembered that the greater part of coronary inflow, unlike flow through other vessels, occurs during physiologic diastole so that it is the aortic diastolic pressure and time which will be the major determinants of coronary flow when the limits of coronary vasodilatation have been reached.

Two exceptions to the above generality do occur and are pertinent to present considerations. These are that more complete extraction of oxygen from blood traversing the myocardium will occur when either anemia or coronary insuffi-

* Data gathered jointly with Dr. Erik Berglund.

ciency is present.[3, 4] It appears, however, that more complete extraction is a second order compensation and will occur when the increased flow is not sufficient to deliver the oxygen required by the myocardium.[3]

THE HEMODYNAMIC CONSEQUENCES OF THE HUFNAGEL OPERATION

The physiologic consequences of the Hufnagel operation[5] may perhaps be more adequately understood with these matters in mind. First of all it is quite likely that the patient with severe aortic regurgitation has an undesirable balance between the augmented work load due to regurgitation, and the limited oxygen available to the myocardium due to the low coronary perfusion pressure during diastole. That is, he has relative coronary insufficiency in the presence or absence of organic coronary artery disease. The frequency with which angina pectoris is a prominent presenting symptom, even in young people, supports this view

The placement of the Hufnagel valve undoubtedly reduces the regurgitant volume (although by how much is not yet clear) and thereby substantially reduces the left ventricular minute work. It is to this that the benefits of the operation must be attributed. At the same time, coronary artery perfusion pressure is almost uniformly decreased. In the tracing published by Rose et al.,[6] end-diastolic brachial artery pressure fell from 53 to 30 mm. Hg, a fall of 43 per cent after the operation, this change was representative of the changes in the 9 patients reported on. In the patient reported on by McKusick et al.[1] in whom pressure tracings were obtained at operation in the ascending aorta before and after the placement of the valve, the findings were in the same direction. End-diastolic aortic pressure above the valve fell from 75 to 35 mm. Hg, a fall of 53 per cent. Perhaps of more significance, mean diastolic pressure fell from 90 to 46 mm Hg, a fall of 49 per cent. The ratio of the time of diastole to the total cycle time fell from 58 to 51 per cent.

It is likely, therefore, that following the Hufnagel procedure there is both a reduced work load and a reduction of the maximum per minute oxygen available to the myocardium. In those patients benefiting from the operation the reduction of work may be thought of as being greater than the functional equivalent reduction of oxygen available to the myocardium. In those not benefiting, the reverse is true. In this sense the operation is a physiologic gamble.

These views are consonant with the finding of Bing et al.[4] in one patient with aortic regurgitation, not in failure, who had a marked increase of coronary flow per 100 gm. Green and Gregg[7] demonstrated that in a dog with severe aortic regurgitation there was a decrease in coronary flow in this instance due to a marked lowering of diastolic pressure.

If the above views are accepted it is clear, as indicated by McKusick et al.,[1] that any attempt to place the valve in the ascending aorta would have dire results in the presence of substantial aortic regurgitation. The short cul-de-sac of aorta proximal to the competent valve, from which the coronaries would expect to derive their perfusion pressure, would have a diastolic pressure very

close to if not identical with that of the ventricle. The placement of the valve nearer the diaphragm would be expected to maintain a higher diastolic pressure above the valve since systolic uptake could then take place in a substantially larger reservoir. The increased regurgitant volume due to the intercostal arteries could be mitigated somewhat by ligation of some of them.

One last point arises out of the above analysis. Although the operative mortality in Hufnagel's hands has been low, others have been discouraged by their early attempts to duplicate his results. A substantial part of the difficulty in the latter instance has arisen immediately following removal of the clamps after insertion of the valve. It can only be guessed at, but it seems reasonable that Hufnagel, after a great deal of practice in the laboratory and extensive clinical experience, completes the procedure in a shorter time than those without this background. At first blush, whether the time of aortic occlusion is three minutes or eight or twelve would appear to have little physiologic significance. It is known, however, that following aortic occlusion there is generalized hypoxia of the tissues supplied by the vascular bed below the point of occlusion and that vasodilatation with a fall in peripheral resistance does occur. The intensity of this might be expected to be some function of the duration of occlusion. Following the placement of the Hufnagel valve, in the presence of aortic regurgitation, the coronary perfusion pressure, as cited above, is substantially lowered per se. If there is added to this a further lowering of coronary perfusion pressure due to widespread peripheral vasodilatation, this might account for the catastrophes encountered at this point of the procedure. The use of a substance such as Aramine[8] injected into the distal aorta might be expected to counteract the diastolic hypotensive state during the post-release period *

RELATIONSHIP BETWEEN POSTOPERATIVE ANEMIA AND "SUDDEN DEATH" FOLLOWING THE HUFNAGEL OPERATION

One important facet of the Hufnagel operation is the so-called "sudden death" that occurs in some of these patients at about the end of the first postoperative week [9]

An attempt will be made to elucidate one of the mechanisms, perhaps the most important one, responsible for this occurrence and suggest a simple remedial measure which might substantially diminish its incidence.

A. CORONARY VASODILATATION AS A COMPENSATORY RESPONSE IN THE PRESENCE OF ANEMIA. As indicated in Fig 1 when the work of the left ventricle is increased, the oxygen supply to the myocardium must also be increased. Recent investigations have more clearly elaborated the effect of anemia on this relationship [3] Figure 2 shows a plot of coronary vascular resistance against coronary perfusion pressure and the changes in this relationship produced by anemia in the dog. It may be concluded that (a) anemia is accompanied by a marked lowering of coronary vascular resistance, and (b) marked degrees of anemia produce a situation wherein the vasodilatory

* One patient with aortic insufficiency in which the Hufnagel operation had been successfully performed several months previously died promptly after the induction of spinal anesthesia

limit is approached and little further decrease in coronary vascular resistance is available. When this occurs a substantial depression of the ventricular function curve is exhibited and a descending limb of the modified Starling or ventricular *function curve also occurs* (Fig 3)

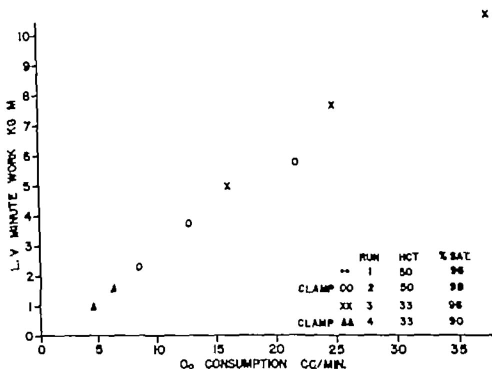


Fig. 1 Relation between left ventricular minute work in kilogram meters and myocardial oxygen consumption in the dog. Four different sets of circumstances were present namely two different hematocrits each with and without coronary stenosis The latter was induced when desired by applying a screw clamp to the tubing feeding the left main coronary artery

Figure 4 shows the magnitude of the increase in flow that it is necessary for the myocardium to acquire in order to maintain any given level of work when varying degrees of anemia are induced. For example, when the left ventricle was performing an external work of 5 kilogram-meters per minute, the left coronary flow was 83 cc. per minute at a hematocrit of 55 At a hematocrit of 32, and at the same work level, left coronary flow was 193 cc. per minute. At a hematocrit of 19, still at the same work level, the left coronary flow was 370 cc. per minute. These changes are even more striking at higher work levels These data are consonant with the clinical findings of Bing et al. who demonstrated an elevated coronary flow per 100 gm. of myocardium in two of the three moderately anemic patients studied.[4]

B THE EFFECT OF CORONARY INSUFFICIENCY UPON THE DEGREE OF ANEMIA WHICH DEPRESSES CARDIAC FUNCTION Figure 5 shows the effect on the canine ventricular function curves of a constant degree of coronary stenosis at hematocrits of 49.5, 33 and then 42.5 At 49 5, coronary stenosis did depress the ventricular function curve and produce a descending limb However, with the same stenosis, at 33 the effect was much more drastic and at 42.5 the effect was intermediate between the two It should be noted that in

the absence of coronary insufficiency, a hematocrit of 33 is readily compensated for (see Fig. 3). Thus, when relative coronary insufficiency is present slight degrees of anemia which are normally well compensated for can substantially depress ventricular function. As mentioned above, there can be little doubt that patients with severe aortic insufficiency have as their fundamental physiologic derangement a ventricular work load which is high in

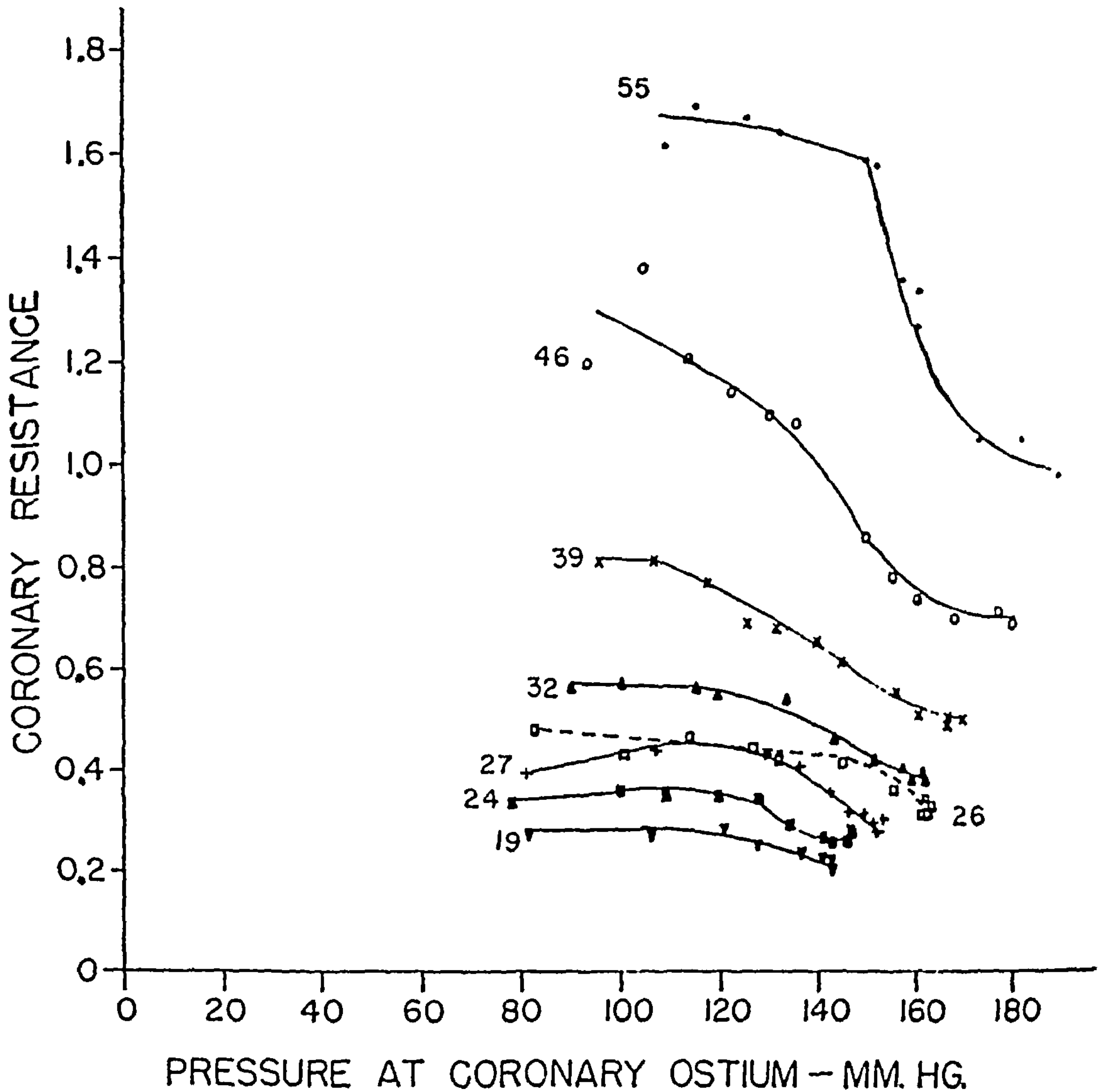


Fig 2. Changes in coronary resistance with anemia. Coronary resistance expressed as mm. Hg pressure drop across the coronary vascular bed divided by the coronary flow in cc /min. Numbers at left of each curve indicate the hematocrit and the dotted line represents the curve obtained after reinfusion of red cells. (After Case, Berglund, and Sarnoff. Am J Med , vol. 18, 1955)

relation to the limited metabolic support of the myocardium, that is, relative coronary insufficiency. *On this basis it is suggested, therefore, that relatively mild degrees of anemia may adversely affect the myocardial function of patients with aortic regurgitation.*

C. POSTOPERATIVE CHANGES IN HEMATOCRIT FOLLOWING THE HUFNAGEL OPERATION. Data are available in the literature on 9 patients as regards hematocrit and blood volume determinations. Table 1, comprised of data

from the work of Rose et al.,[6] shows these changes Hematocrits fell from an average of 41.5 to 34.8 when taken an average of 24.2 days (14 to 36 days) after the Hufnagel operation. All 9 patients showed a decrease. These varied from 3 to 16 hematocrit points

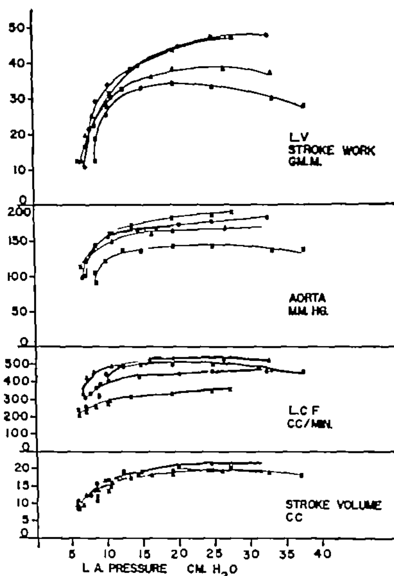


Fig. 3 Ventricular function curves in anemia. At the top the left ventricular work is plotted against mean left atrial pressure. Also shown are aortic pressure, left main coronary artery flow, and stroke volume plotted against mean left atrial pressure. ××× = hematocrit of 43%. ooo = 32%. ΔΔΔ = 24%. □□□ = 17.5%. Note the depression of the ventricular function curve at 24% and the further depression and the descending limb at 17.5%. (After Case, Berglund and Sarnoff: Am. J Med.)

Several things are interesting in regard to this group First, as will be shown below (Fig 6), 24 days after the onset of red cell destruction due to the insertion of a ball valve prosthesis, increased erythropoietic activity has already set in and the hematocrit has begun to return to its preoperative level, at least in the dog. It is likely, therefore, that the postoperative hematocrits shown in Table 1 are not the lowest exhibited by these patients.[11] Second, as concomitant blood volume studies were done it is possible to eliminate

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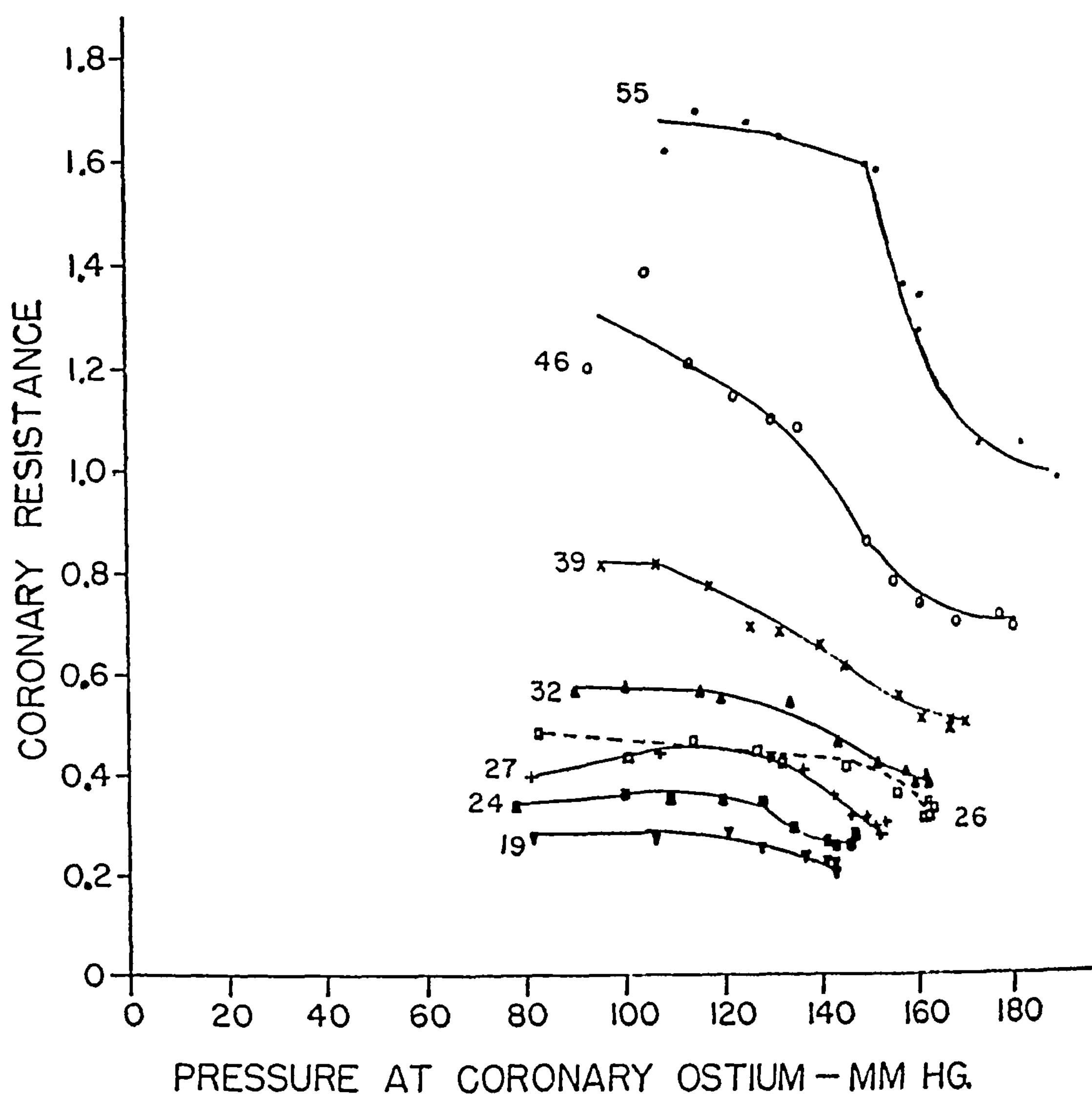


Fig 2 Changes in coronary resistance with anemia. Coronary resistance expressed as mm. Hg pressure drop across the coronary vascular bed divided by the coronary flow in cc /min. Numbers at left of each curve indicate the hematocrit and the dotted line represents the curve obtained after reinfusion of red cells (After Case, Berglund, and Sarnoff Am J Med , vol. 18, 1955.)

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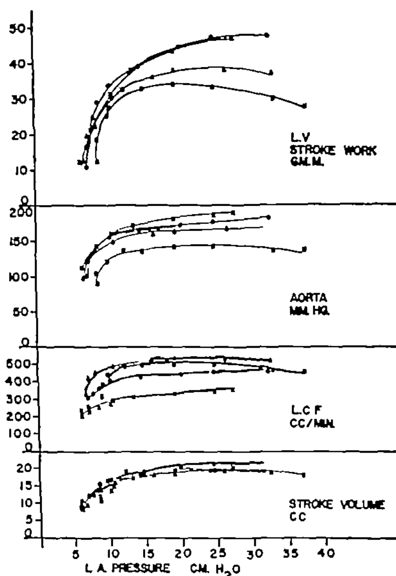


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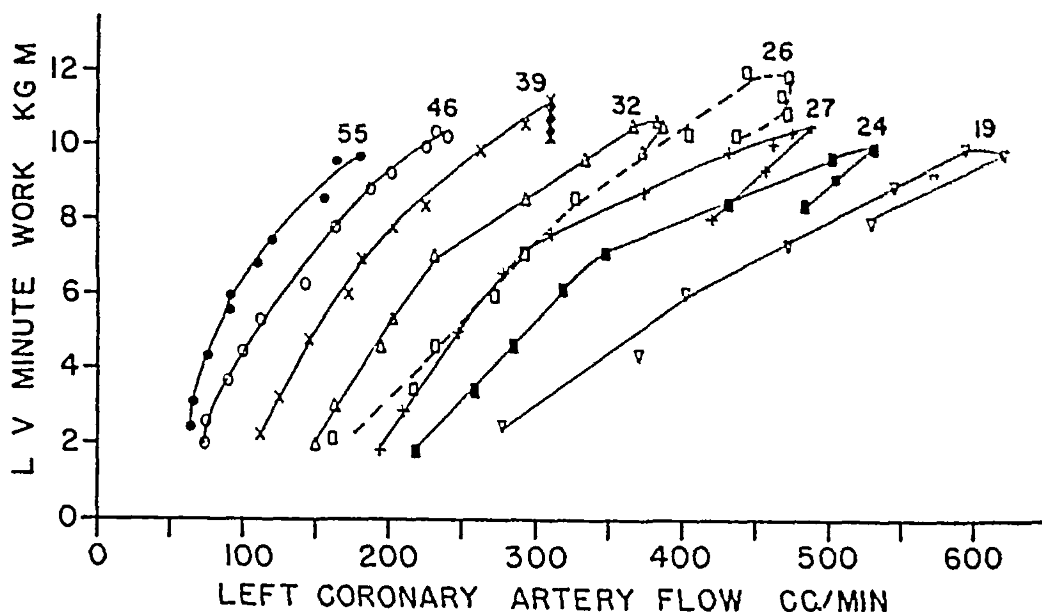


Fig 4 Work-flow relationships during anemia Left ventricular work per minute in kg -m is plotted against left coronary flow in cc /min. Figures above each curve represent the hematocrit The dotted line is the curve obtained after the reinfusion of red cells. (After Case, Berglund, and Sarnoff. Am. J Med., vol. 18, 1955.)

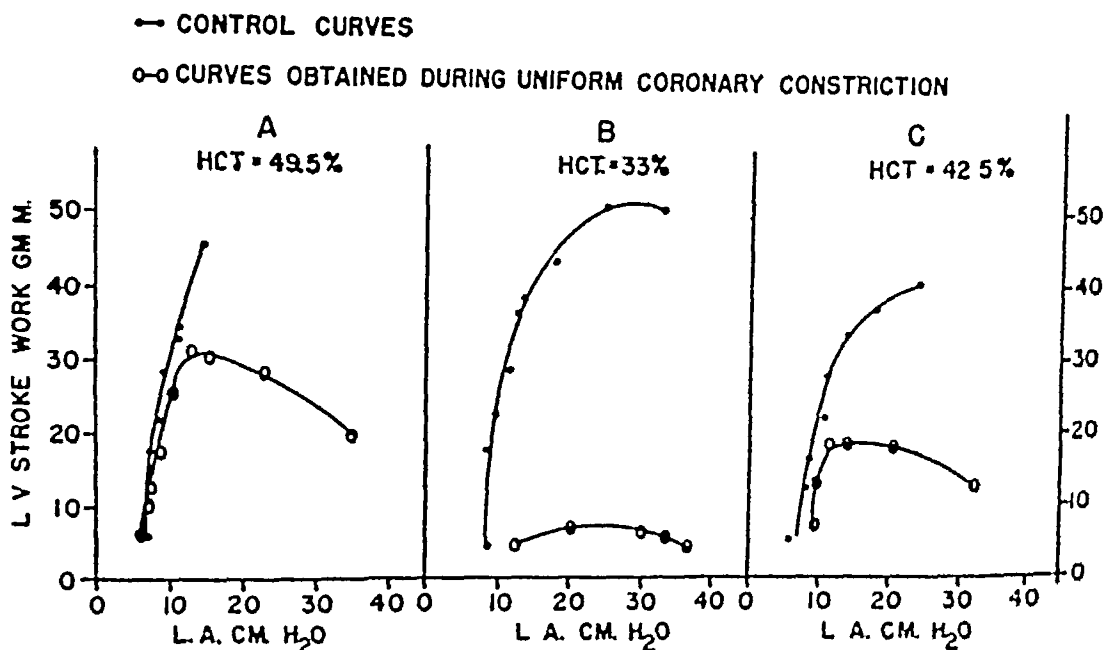


Fig. 5 Effect of coronary stenosis and anemia on ventricular function curves. Dots represent points from control curves Circles represent points obtained during application of a uniform coronary constriction. Dog was made anemic between A and B, and red cells were reinfused between B and C. (After Case, Berglund, and Sarnoff. Am J Med , vol. 18, 1955)

hemodilution as a reason for the uniformly lower postoperative hematocrits Third, none of these 9 patients exhibited "sudden death" in the postoperative period, the earliest death being at four months. It would, of course, be interesting to know the hematocrit changes in those patients exhibiting this syn-

drome, although it is to be emphasized that a quite mild anemia may leave one patient relatively unaffected while having a lethal effect on another As shown above, the effect of any given degree of anemia in depressing myocardial function depends upon the intensity of the relative coronary insufficiency already present. Lastly, it appears unlikely that the postoperative fall in hematocrit is due to surgery alone.

TABLE 1

Patient	*Days**	*Arterial hematocrit (%)*	*Total blood volume (L)*
M. H.	1 pre	40	4.08
	20 post	32	3 75
W A.	8 pre	39	2.73
	36 post	33	2.61
C. F	4 pre	43	4.06
	14 post	35	4.34
H. S	7 pre	39	7 14
	18 post	36	6.47
S R.	3 pre	37	3 60
	21 post	33	4.33
L. F	1 pre	44	—
	21 post	40	4.92
W J	3 pre	48	5.50
	28 post	32	4.76
G P	9 pre	43	5 09
	30 post	38	5.85
T D	26 pre	40	5 47
	30 post	34	4.36

* pre = Number of days before operation that studies were performed
post = Number of days after operation that studies were performed
Modified from J C. Rose and others: J Clin. Investigation, 33:891, 1954.

The fact that anemia of varying degrees may occur in patients after the Hufnagel operation can, of itself, in no wise be construed as a contraindication to the operation, since the anemia is remediable. What does stand out, however, is that the hematocrit should be followed closely, especially in the immediate postoperative period, in order to relieve the coronary vascular bed of the necessity of dilating in response to anemia, it has, in all likelihood, already extended itself maximally in this direction.

Whether it should eventually turn out that the postoperative fall in hematocrit is due to hemodilution, the surgery itself, or, as suggested below, increased red cell destruction due to the valve, is of no immediate practical consequence. The myocardium cannot discriminate between the causes of anemia, it senses only the result. Its main biologic need is to acquire sufficient oxygen to sustain the work required of it. Since, in aortic regurgitation, flow is limited by a low diastolic coronary perfusion pressure, this need will be more readily met when the oxygen-carrying capacity of each 100 cc. of blood is not limited

by a low hematocrit. This may be especially true after the insertion of the Hufnagel valve when aortic arch diastolic pressures are further reduced [1,6]

D. RED CELL DESTRUCTION PRODUCED BY THE LUCITE BALL VALVE PROSTHESIS. Attempts have been made to circumvent the aortic valve as a

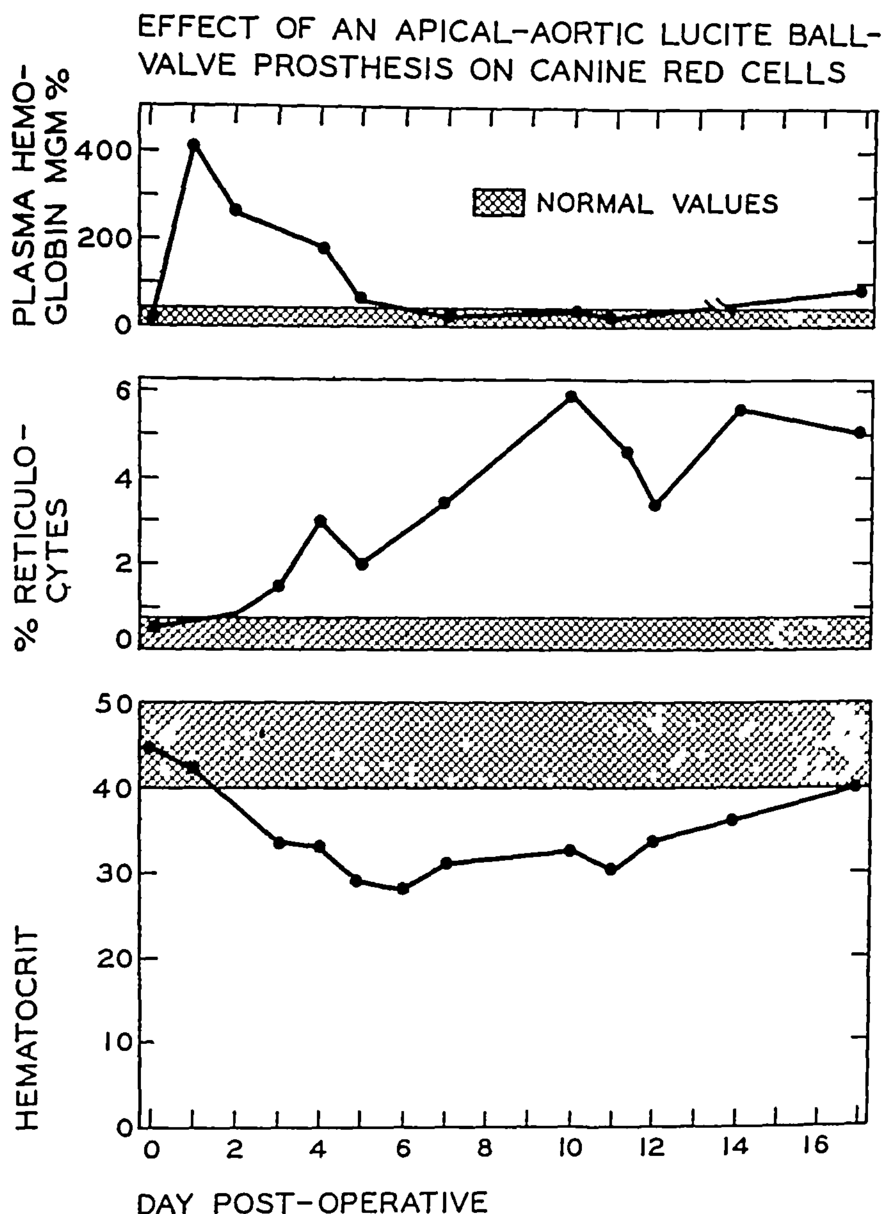


Fig 6 Intravascular hemolysis, anemia and reticulocytosis following the insertion of a Lucite ball valve prosthesis between the left ventricular apex and thoracic aorta in the dog Ascending aorta occluded

means of alleviating aortic stenosis and possibly also aortic insufficiency (apical-aortic anastomosis). A résumé of these efforts was given earlier in this volume and the details have been published elsewhere.[10] Briefly, the method consists of inserting into the apex of the dog's left ventricle a Lucite tube which leads blood through a Hufnagel Lucite ball valve and then into a T tube placed in the thoracic aorta. Thereafter, the ascending aorta is completely occluded It was noted early in these experiments that hemo-

globinuria and hemoglobinemia were a frequent if not constant postoperative finding.[10] Since Hufnagel had already had such valves in dogs for long periods of time without observing this, it was felt that the valve was innocent and other factors were sought, unsuccessfully. However, no hematologic data have been reported regarding dogs with Hufnagel valves in the Hufnagel position and with aortic insufficiency. These data are currently being obtained.

Figure 6 shows the changes in plasma hemoglobin, reticulocyte count and hematocrit in the first seventeen days after the insertion of a ball valve prosthesis in the apical-aortic position in the dog. Plasma hemoglobin rose from 8 to over 400 mg. per 100 cc. on the first postoperative day and thereafter declined. The hematocrit reached its low point on the sixth day and thereafter gradually returned toward its previous level as the reticulocyte count rose. Of interest in regard to the "sudden death" syndrome observed after the Hufnagel operation is the observed time of the lowest hematocrit, namely, the sixth day. Of further practical importance is the apparent ability of the erythropoietic system to compensate for the increased rate of red cell destruction. The initial peak in the plasma hemoglobin curve suggests that the ball valve initially destroys the more fragile members of the red cell population. Whether this variation is simply a normal distribution in red cell fragility or is a function of cell age or both is not as yet known. A straight Lucite tube in the aorta alone did not cause a postoperative elevation of plasma hemoglobin or anemia in the 4 dogs so studied.

Data to be reported on elsewhere[11] deal with the survival of Cr^{51} tagged red cells when given to dogs with the Lucite ball valve in the apical aortic position. These studies reveal that apparent cell half-time is shortened to 3 to 10 days as compared to the 21 to 30 days found in normal controls. Similar studies with the Lucite ball valve in the Hufnagel position in dogs with aortic insufficiency have been undertaken.

Detailed and definitive data are not available on the relative mechanical fragility of the red cells of man and dog. However, when one exerts undue pressure in withdrawing a blood sample from a dog, hemolysis frequently ensues, whereas this is rare in man. Osmotic fragility is somewhat higher in the dog but it is to be emphasized that osmotic fragility and mechanical fragility are not comparable. Studies comparing the mechanical fragility of canine and human red cells in a standard pumping chamber with and without a Hufnagel valve are being undertaken and will be reported elsewhere.

GENERAL CONSIDERATIONS

The above considerations do not reflect the substantial advances made by Hufnagel in this field. The fact remains that he has devised and demonstrated a reasonably safe means of valving circulatory currents. Further, it is entirely likely that the severely ill group of patients he has operated on are, as a group, better off than would have been the case without his operation. However, as has been suggested,[10] the physiologic gamble could be converted to a certainty by closing or tightly stenosing the aortic valve proximal to the coronary ostia and providing an alternative outflow path with a competent

valve. Under these circumstances regurgitation would be completely abolished and coronary perfusion pressure maintained.

SUMMARY

1. Data are presented which support the view that an increased ventricular work load is accompanied by an increased myocardial oxygen requirement.
2. The fundamental physiologic derangement in the patient with aortic regurgitation is an increased left ventricular work load in the presence of a lowered reserve of myocardial oxygen availability The latter is occasioned by the low aortic diastolic or coronary perfusion pressure. Following the Hufnagel operation, the left ventricular work load is reduced by an unknown amount and the diastolic coronary perfusion pressure is approximately halved. It is suggested that in this sense the operation is a physiologic gamble.
3. An attempt has been made to associate the postoperative anemia seen after the Hufnagel operation with the "sudden death" syndrome occasionally seen in the first four to eight days after it.
4. It was demonstrated that one substantial component of the circulatory response to anemia is a decrease in coronary vascular resistance. When the limits of this decrease in resistance are approached, myocardial function is depressed.
5. When coronary insufficiency is present degrees of anemia which are normally well tolerated will substantially depress ventricular function.
6 It is believed that patients with aortic regurgitation have relative coronary insufficiency and therefore that relatively mild degrees of anemia will affect them adversely.
7. Intravascular hemolysis and anemia have been demonstrated following the insertion of a Lucite ball valve prosthesis in the apical-aortic anastomosis position in the dog. The hematocrit frequently reaches its lowest point on the sixth to the eighth postoperative day.
8. Intravascular hemolysis following the insertion of the Lucite ball valve in man in the Hufnagel position has not as yet been looked for. Whether or not it is responsible for the postoperative anemia, the latter should be promptly corrected and this matter is of greatest import in the early postoperative period.

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DISCUSSION

Charles P Bailey (*Philadelphia*)

I am greatly impressed by this work by Dr Hufnagel, and if it has seemed that we thought there might be another approach to aortic insufficiency, I am sure you will all understand that we are all trying to make progress

I do think that what Dr Sarnoff has just said about the fact that there is coronary insufficiency in these cases, and that it may possibly be aggravated in borderline individuals by the placement of the valve beyond the left subclavian artery, is quite important.

In our very small series of cases the diastolic pressure in the upper extremities fell right after surgery and stayed lower than preoperatively, suggesting that there was less perfusion pressure as indicated. I think Dr Hufnagel probably would like to comment on that.

CONCLUDING REMARKS

Charles A. Hufnagel (*Washington, D C*)

I am sure that in the early cases we had problems which arose from allowing the patient to maintain too low a hematocrit value. It was difficult for us to persuade our medical colleagues that the transfusion of cells in this early phase was good for a heart that was already in such serious trouble. I am sure that they are now well convinced, and that this problem is distinctly less important.

As time has gone on, we have taken cases when they are just beginning to have trouble, and the over-all results have strikingly improved.

We have not been particularly interested in putting this valve in the ascending aorta, for reasons which are quite apparent in relation to coronary flow—that unless one maintains the run-off from the cerebral vessels and the arm

PANEL DISCUSSION ON INTERATRIA SEPTAL DEFECTS

HARRIS B. SHUMACKER (*Indianapolis*), MODERATOR
CHARLES P. BAILEY (*Philadelphia*)
S. GILBERT BLOUNT, JR. (*Denver*)
CLARENCE CRAFOORD (*Stockholm*)
JOHN W. KIRKLIN (*Rochester, Minn.*)
CONRAD R. LAM (*Detroit*)
ANDREW G. MORROW (*Bethesda, Md.*)
TYGE SONDERGAARD (*Aarhus, Denmark*)

DR. BLOUNT

I find myself the only internist among seven surgeons I am allowe minutes to discuss diagnosis and the selection of patients.

In patients above the age of two years, the diagnosis on clinical g alone is relatively easy.

The diagnosis in infancy is an entirely different story. The histor little help in the diagnosis. It is the history of any patient with a left t shunt, and, depending upon the volume of the shunt, the symptoma will vary

The *physical examination* is of extreme importance. These patients n underdeveloped. They are usually not cyanotic. Only those who have plications and who develop pulmonary hypertension have cyanosis.

When one examines the chest by inspection and palpation, one u finds that there is a systolic pulsation in the second left intercostal Usually there is no shock. There is a hyperdynamic ventricular beat more like a left ventricular beat than a right ventricular beat, becaus the atrial septal defect we have the primary example of the so-called di overload of the right ventricle With almost all other kinds of cong heart disease the right ventricle is in systolic overload and is a predomi hypertrophied rather than dilated ventricle

Auscultation is of help The murmur is usually not a significant on oftentimes it may be passed as an innocent murmur. It is usually sol blowing in the second intercostal space, and rarely is accompanied by a The second heart sound over this area may show reduplication, bu increase in intensity, because the usual atrial septal defect has a rela normal pulmonary diastolic pressure

The *electrocardiogram* is of great importance. It shows in a classic m the so-called diastolic overload of the right ventricle, that is, the patt

partial right bundle branch block. When we have beginning pulmonary hypertension, one notes a change in the electrocardiogram to the picture of a systolic overload.

Fluoroscopy is also important. The vascularity of the lung fields is increased. The main right and left pulmonary arteries are increased in size, and apparently the amplitude of pulsations is increased, although I find it difficult to differentiate between actual expansile pulsations of the pulmonary tree.

If one gets a good-sized vessel on end and sees it expand, it is very helpful. The heart, of course, usually is considerably enlarged. The right atrium, it is important to note, is particularly enlarged, and also the right ventricle The left side of the heart including the aorta is usually within normal limits

The passage of the catheter across into the left atrium, and the finding of a left to right shunt at the atrial level, are of extreme importance in making this diagnosis We have had very little experience with differential dye curves and the injection of dye into the right and left pulmonary arteries, apparently these methods are of help in differentiating atrial septal defects from anomalous pulmonary venous connections

With our equipment angiocardiography is of no help, although I know that with faster speed it is of considerable help and can show the atrial defect very nicely

In the selection of cases, we feel that the patient should have a very significant left to right shunt, and we have arbitrarily set the figure as a pulmonary index three times the systemic. We realize that this is arbitrary and will change.

As far as pulmonary hypertension is concerned, I think it is important to differentiate the pulmonary hypertension secondary to flow and minimal changes in the pulmonary vascular bed from the pulmonary hypertension of marked increase in pulmonary vascular resistance with small left to right shunts Thus, if we have a patient with a relatively high pulmonary artery pressure, such as 100/40, but there is a large left to right shunt, then we feel that this patient certainly needs operation.

If there is cyanosis, I know there will be some difference of opinion. The patient who has cyanosis with a right to left shunt and no increase in pulmonary flow, is beyond help as far as operation is concerned. Certainly if one does attempt operation he must realize that the risk is very high.

I believe we all will see the time when the atrial septal defect will be closed just as the patent ductus is today It is a single defect which lends itself readily to correction.

Dr. Morrow

In a symptomatic patient with an atrial septal defect and a large left to right shunt, routine cardiac catheterization will most often lead to the correct diagnosis When, however, the defect and shunt are small or there is an associated valvular lesion or interventricular defect, the usual techniques may fail to reveal the interatrial defect or yield only suggestive evidence as to its presence. Accordingly, we have been evaluating two ancillary methods for the diagnosis of interatrial defect.

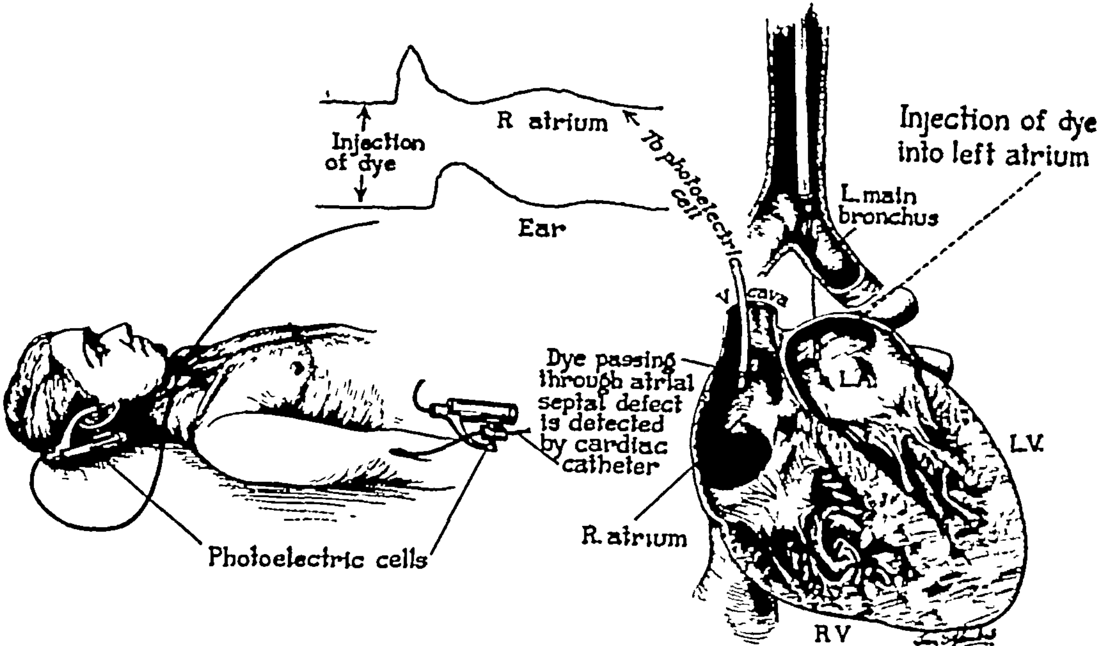


Fig. 1 Transbronchial injection of Evans blue dye into the left atrium and method of determination of appearance time in the right atrium and ear blood

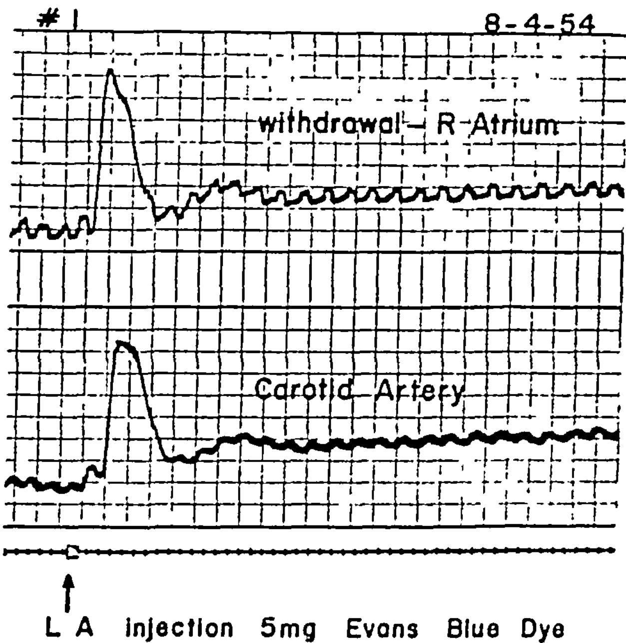


Fig 2. Record of appearance of dye in the right atrium and carotid artery in experimental animal with interatrial defect The appearance of dye in the right atrium before the appearance in the carotid artery is diagnostic of a shunt

The first is illustrated in Fig. 1. It consists of the transbronchial injection of Evans blue dye into the left atrium as blood is being continually sampled from the right atrium by a cardiac catheter. Densitometers on the catheter and the patient's ear record the passage of the dye through the circulation. If, as in Fig. 2, dye is found in the right atrium before its appearance at the

ear, the presence of a shunt is established. In the absence of a shunt or when blood is withdrawn proximal to the defect (Fig 3), dye appears on the right only *after* its passage through the peripheral artery The method has been used in 4 patients with the demonstration of two interatrial and two interventricular defects Unfortunately, it does not seem applicable in children and of course requires familiarity with the technique of left atrial puncture.

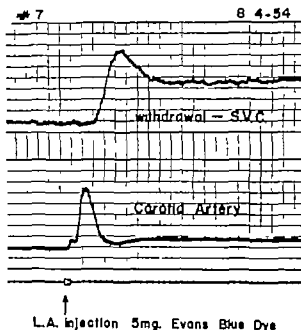


Fig. 3 Experiments similar to that of Fig. 2 but blood is sampled from the superior vena cava rather than right atrium. Dye appears in the carotid artery before it does in the venous blood.

A second method, devised by Dr James Callaway, has been applied in 40 patients It depends upon the partition of nitrous oxide between the arterial and venous circulations A double lumen catheter is passed with the proximal lumen in the superior or inferior cava and the tip in the right atrium. Simultaneous samples are drawn from these sites and from the femoral artery over a period of one minute, first while the patient breathes room air and then while he breathes 15 per cent nitrous oxide. The samples are analyzed for nitrous oxide and oxygen content in the Van Slyke apparatus. In the absence of a shunt (Fig 4) extremely small amounts of nitrous oxide are present in the venous blood while the arterial level is 3 to 5 volumes per cent. Since the ratio of the arteriovenous difference to arterial level is much higher for nitrous oxide than for oxygen, shunts are more easily demonstrated. This is illustrated in Fig. 5 where the results are expressed as the ratio $\frac{\text{RA-SVC}}{\text{FA-SVC}}$ In Fig. 6 are the results of both oxygen and nitrous oxide methods in 18 dogs with and without atrial defect. The oxygen samples were also drawn

NORMAL

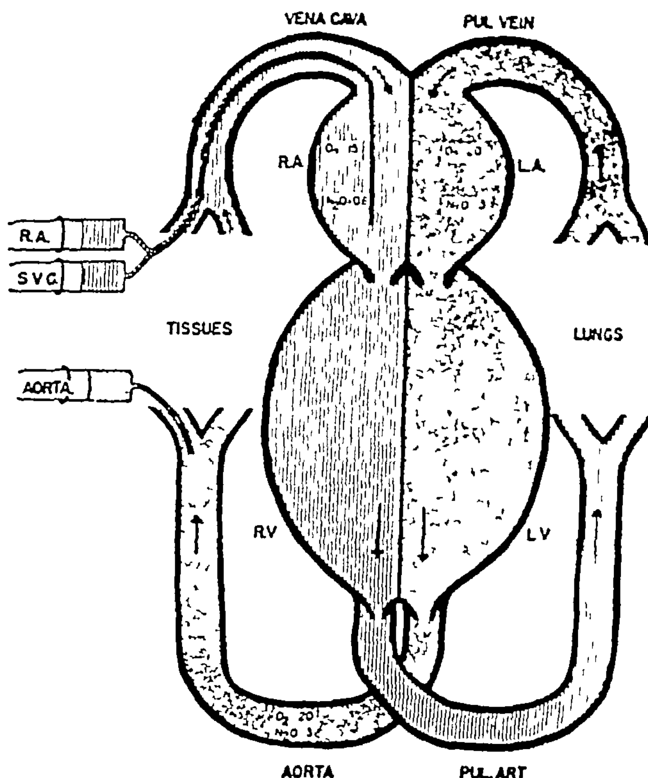


Fig 4. Partition of oxygen and nitrous oxide in a normal subject breathing 15 per cent nitrous oxide for one minute.

INTER-ATRIAL SEPTAL DEFECT

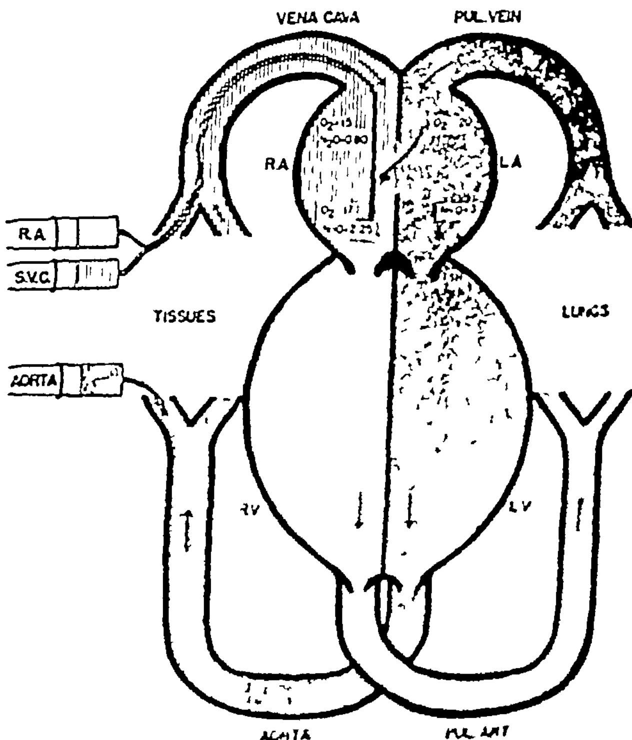


Fig 5. Partition of oxygen and nitrous oxide in a patient with interatrial septal defect See text.

simultaneously with the double lumen catheter From these and similar data we have concluded that if the ratio $\frac{\text{RA-SVC}}{\text{FA-SVC}}$ exceeds 0 32 it is diagnostic of a shunt. Ratios lower than 0.32 may include small shunts, but no normal animals or patients have had higher values

Comparison of O_2 and N_2O in Demonstration of Atrial Septal Defects

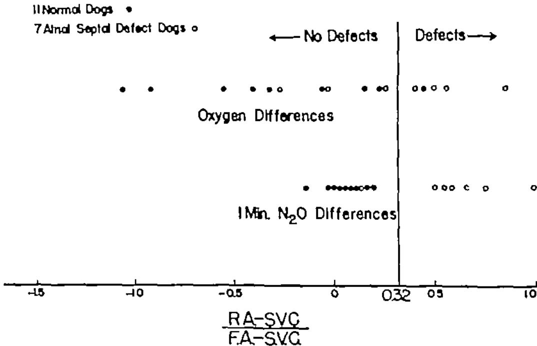


Fig 6. Oxygen and nitrous oxide values determined in experimental animals with and without interatrial septal defects. See text.

	O_2 CONTENT			N_2O CONTENT
S.V.C.	7 5	RA	8.9	I.V.C. 02
	7 7		9 0	
I VC	9.8		9 5	R.A. 1 81
	7 6		9.3	
	7 6	simultaneous	8 8	F.A. 4 08 (1 min. integrated samples)
mean	8 0		9 1	
	F.A. 11 9			

$$\frac{9 1-8 0}{11 9-8 0}=0 28 \qquad \frac{1 81-02}{4 08-02}=0 44$$

Fig. 7 Chart illustrating the greater sensitivity of the nitrous oxide partition test as compared with that of oxygen in a child known to have an interatrial septal defect.

content, and then draw the catheter back slowly and come into a low pressure area with a low oxygen content, it is highly suspicious

The important thing is that it is not the position of the defect that is in any way helpful in making a differential diagnosis, but it is the attending defect of the atrioventricular valves that is diagnostic.

We have had one case in which there was a so-called ostium primum type defect but the atrioventricular valves were normal, certainly with no insufficiency, and it portrayed a picture that was indistinguishable from the secundum type of defect. However, I think there are some findings that do suggest the diagnosis

DR. SHUMACKER

From our experience in Indianapolis, there is plenty of room for improvement in the recognition of defects of the atrial septum in infants It is with this group that we have had our largest percentage of diagnostic errors They have included cases of simple patent foramen ovale with an associated ventricular septal defect, instances of atrioventricularis communis, and a case with patent foramen ovale, patent ductus arteriosus and a stenotic bicuspid aortic valve. We have had this difficulty in spite of the very serious efforts on the part of our pediatric colleagues

Dr Crafoord, would you say a few words about your efforts in Stockholm to estimate the size and site of the defect in the atrial septum?

DR. CRAFOORD

I am in full agreement with Dr Taussig's earlier statement that it is extremely important to have as perfect a diagnosis in any case that is to be taken into the surgical operating room as can be attained.

Our general effort in Stockholm has been to try to visualize the anatomy of the different defects as close to reality as possible. In this effort, one of our group has constructed a single or double balloon catheter The balloons, which are close to the catheter when they are not inflated, can be passed with the catheter from the right atrium to the left. By injecting contrast media in the most peripheral balloon, or into the only balloon present if it is a single balloon catheter, and gradually trying to take the catheter back, one can exactly determine at which size of the balloon it is no longer possible to get it back from the left atrium over to the right. By that method the size of the defect can be determined.

Also, the site can be determined if we compare the roentgenograms when the balloon is inflated, with the angiocardiograms. We can also determine about where the lower level of the defect is located, and can get a fairly good amount of information as to whether we are dealing with a septum primum or septum secundum defect.

It also helps to differentiate between a foramen ovale and an abnormal venous return, an atrial septal defect combined with a normal venous return, and a foramen ovale and normal venous return. If we blow up first the peripheral and then the nearer balloon, we can block the defect completely If there is still an admixture of oxygenated blood into the right atrium, we have

demonstrated that we have an abnormal venous return. If the balloon is only slightly inflated and it is impossible to take it back again, then we have demonstrated that it is not a real defect and we have a patent foramen ovale. By this method we have obtained much useful information in our preoperative studies.

DR. SHUMACKER

Perhaps now it would be a good idea to move on to a discussion of some aspects of treatment itself. I will ask Dr. Bailey to continue. Dr. Bailey is the originator of one very useful and popular method of closure of atrial septal defects.

DR. BAILEY

Basically, in these cases you have a lack of cardiovascular tissue in the septum expressed by the defect. There is a greatly dilated right atrium in all cases which have a left to right shunt, and so you have an excess of cardiovascular tissue in the form of the distended right atrial wall. If you use it

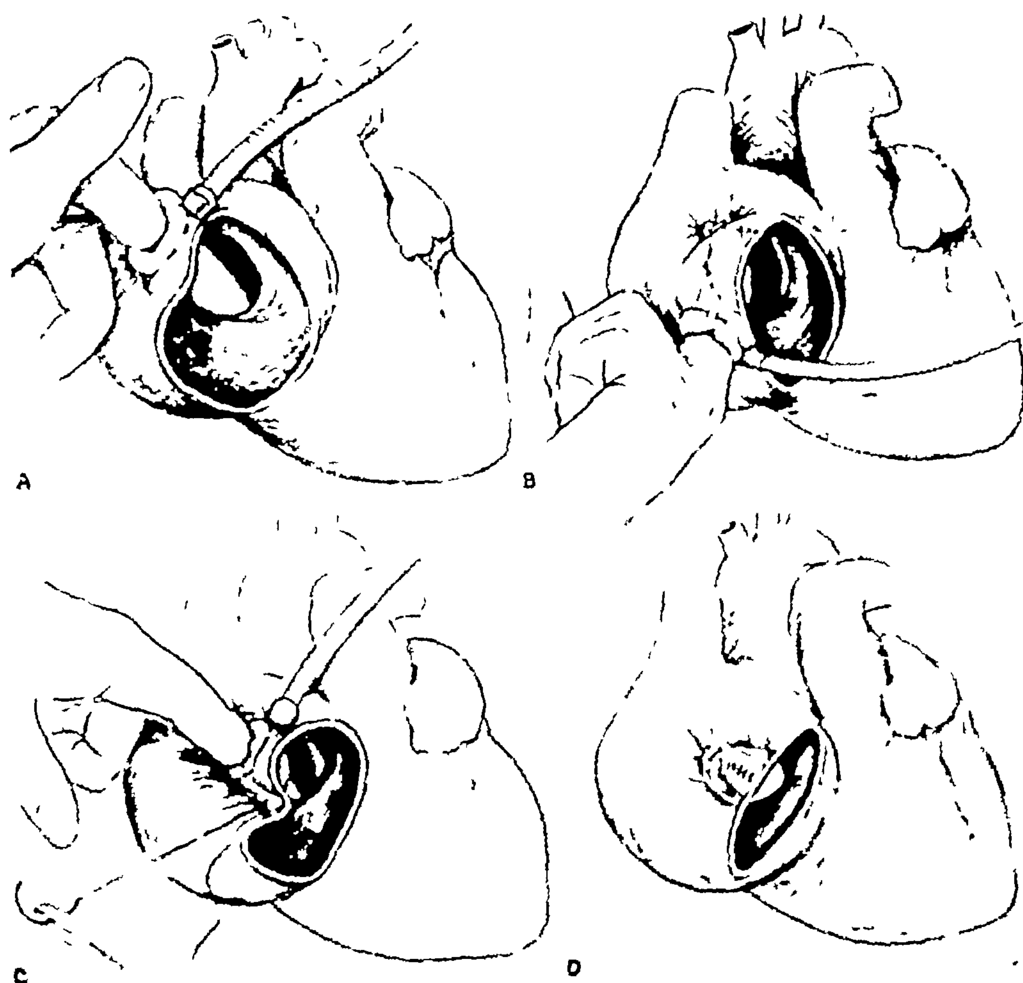


Fig. 1 *A*, Digital exploration of septal defect via right auricular appendage *B*, Placement of first suture in atrioseptopexy *C*, Progressive approximation of invaginated right atrial wall to periphery of septal defect *D*, Completion of atrioseptopexy. (From Bailey and others Journal of Thoracic Surgery, vol. 26, 1953 ,

economically you can utilize the excess to cover the defect, the finger in the heart guiding the sutures

In Fig. 1*C* the sutures are being placed, gradually encircling the entire defect. If it is a large defect, of course, you cannot completely encircle it, but you *may at least* exclude the anterior portion of the atrium from the vena caval flow, using all of that portion of the atrial wall to cover the large defect here. The vena caval blood is directed posterior to the defect by way of a newly created intracardiac channel.

Figure 2 shows the first patient operated on by this technique. She is lying on her back. One sees the remainder of a formerly greatly dilated right atrium

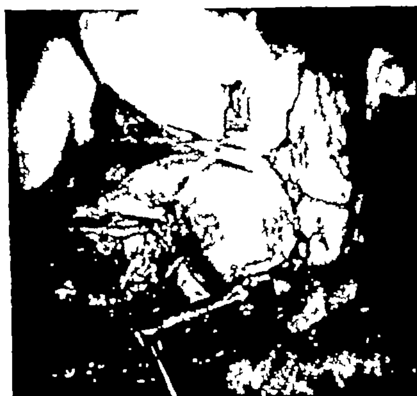


Fig. 2 Photograph of the heart after completion of the first successful atrioseptopexy (From Bailey· Surgery of the Heart. Courtesy of Lea & Febiger.)

Multiple sutures have been placed to tack down all of the anterior portion of the atrial wall to cover the defect. This operation was performed on January 11, 1952. She has made a nice recovery In Table 1 you can see the systemic blood flow of 4 liters per minute, and pulmonary blood flow of 17.3 liters per minute both become altered to 5 9, the left to right shunt having been abolished. I believe that this is the first physiologically proven case of closure of a septal defect.

We have run into various complications and problems which may be associated with these lesions, such as anomalous drainage of the right pulmonary veins into the right atrium In these individuals the septal defect usually is located posteriorly and close to the mouths of the anomalous veins I suppose this association may be related to excessive trans-septal flow during the period of development of the interatrial septum.

TABLE 1 CATHETERIZATION DATA IN A CASE OF ATRIAL SEPTAL DEFECT BEFORE AND AFTER CLOSURE BY ATRIOSEPTOPEXY

	Preop.	*Postop.* 17 Days	3 Mos.
PVC (mm. Hg)		8	
PA (mm Hg)	90/40 (65)	48/33 (40)	
RV (mm Hg)	90/10 (55)	48/0 (22)	
RA (mm. Hg)	10/2 (5)	10/2 (5)	
BA Sat'n (%)	94 0	82.4	95 0
O_2 Cons. (cc /min)	190	177	
SBF (L/m)	4 1	5 9	
PBF (L/m)	17.3	5.9	
Left to right shunt (L)	13 2	0	

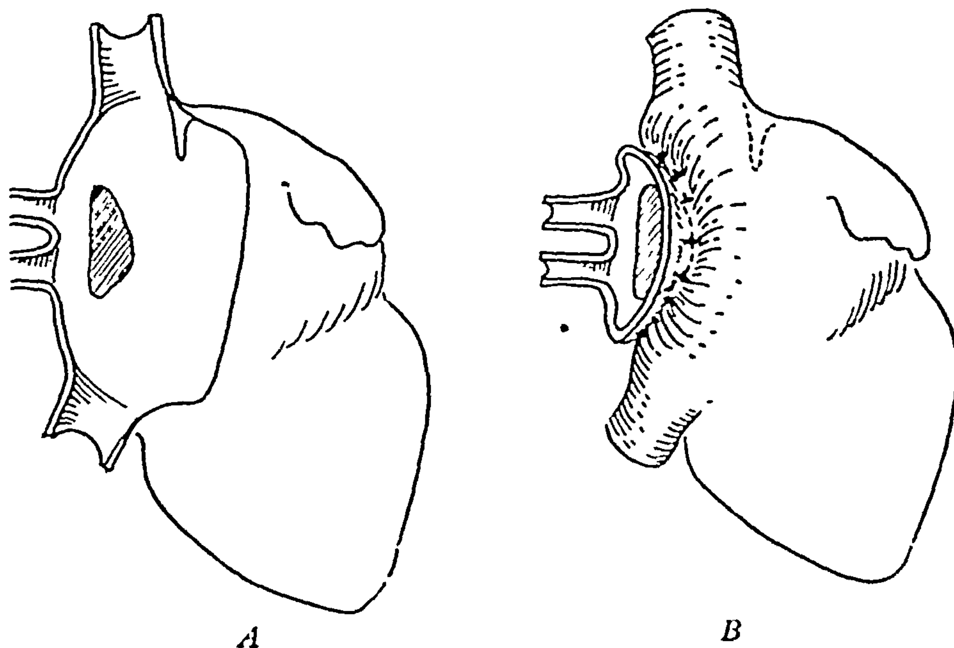


Fig 3 *A*, Single anomalous right pulmonary vein associated with an interatrial septal defect. *B*, Anomalous vein retransposed through defect and latter functionally closed.

This abnormal drainage can readily be corrected by the technical modification, in which you suture the lateral wall of the atrium to the anterior edge of the defect, continuing the suture line posteriorly above and below. Then the blood flow is directed from the pulmonary veins through the defect into the left atrium. I must say this method was suggested to me by one of my residents, Dr. Wilford Neptune, who is now in Boston at the Overholt Clinic

Modifications of this technique, when one pulmonary vein empties into the superior vena cava, may be made in this fashion, or by re-routing one vein down into the other and then doing the operation as indicated.

There are many different types of anomalous pulmonary venous drainage, and we cannot go into all of them at this moment

We have run into 3 cases of septal defect with coexistent mitral stenosis You see you can operate right through the defect on the stenotic valve, either with a knife or a finger After you have repaired the defect both elements causative of disability have been overcome.

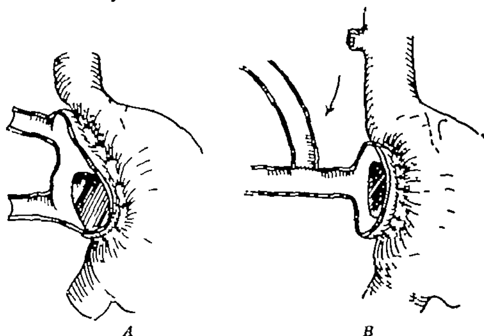


Fig. 4. *A* If the superior pulmonary vein drains into the superior vena cava close to its terminus, the suture line may be extended obliquely up the superior vena cava to effectually separate the two venous circulations *B*, If the upper pul monary vein inserts into the superior vena cava at too high a level for this procedure, it may be divided and reanastomosed in end to side fashion to the lower one which will be treated as described. (From Bailey· Surgery of the Heart. Courtesy of Lea & Febiger)

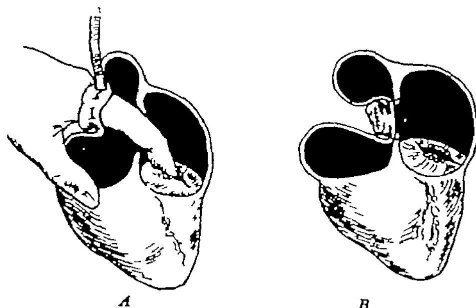


Fig. 5 *A* Performance of digital mitral commissurotomy via right auricular appendage and the septal defect. Incisional commissurotomy may be similarly accomplished. *B*, Closure of septal defect to complete cure. (From Bailey and others: Journal of Thoracic Surgery vol. 26, 1953)

At this time the number of secundum cases which we have operated on is close to 50, and the mortality is below 9 per cent at this moment. The clinical improvement is impressive. We have catheterized 11 cases postoperatively and have heard from some of these patients in far-away cities. To date there are apparently only 2 in whom we did not accomplish complete closure of the defect.

TABLE 2. TREATMENT OF OSTIUM SECUNDUM DEFECT

	No. of Cases	*Percentage*
Total Cases	31	
Associated mitral stenosis	3	9.7
Associated anomalous pulmonary venous drainage	5	16.1
Mortality	3	9 7
Clinical improvement in surviving patients	27	100
Postoperative cardiac catheterization	11	39 3
Complete abolition of shunt in those catheterized	9	81.8
Great reduction of shunt in those catheterized	2	18 2

TABLE 3 TREATMENT OF OSTIUM PRIMUM DEFECTS

	No. of Cases	*Percentage*
Total Cases	16	
Associated mitral stenosis	0	—
Associated anomalous pulmonary venous drainage	2	12.5
Mortality	11	68.7
Clinical improvement in surviving patients	5	100
Postoperative cardiac catheterization	2	40
Complete abolition of shunt in those catheterized	2	100

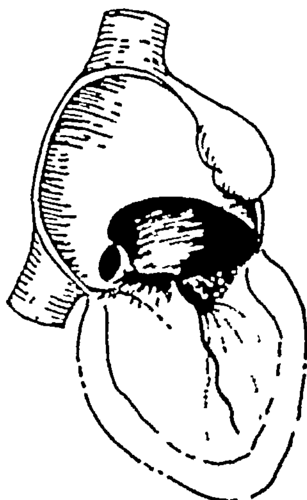


Fig 6 Diagram of ostium primum defect which demonstrates the anatomic problem of surgical repair

We have operated upon 16 patients with ostium primum defects, in whom we had to sew into the interventricular septum—at least that was the intention—and we have had a mortality in 11 cases. That is a pretty high mortality. Three of the deaths were due to the inadvertent production of a complete heart block. That was a 75 per cent mortality in that group. The last

two operated on got along very well because no attempt was made at repair

Figure 6 shows the reason why we don't think atrioseptopexy is a good procedure in patients with persistent ostium primum. You have to sew into the upper portion of the interventricular septum, and in the absence of atrial tissue in this region the common conduction bundle must lie right on top of this interventricular septum, probably a little to the right side. It would seem nearly impossible to avoid picking it up when suturing in this region. Patients do not do well when the bundle of His is interrupted. We are inclined to think that another technique of management for this type of defect is desirable.

DR. SHUMACKER

Dr Lam, we understand that you have an innovation in the surgical management of this condition, and we would like to hear about it.

DR. LAM

Our cases have fallen rather easily into two groups, as distinct from each other as black from white. In our series of 16 cases, 12 were very easily repaired by the method which I shall describe, while 4 (instances of the so-called ostium primum defects) could not be closed even partially Two of the latter group died following exploration, there was no mortality in the former group

If one will examine an autopsy specimen of a heart with interatrial defect of the operable type, it will be seen that dislocation of the posterior rim of the defect forward and to the left will completely close the opening, regardless of its size. A row of sutures maintaining this position would accomplish the desired result.

The method which we use now is as follows.

The heart is exposed through a generous antero-lateral incision through the fifth interspace on the right, and the pericardium is opened widely The Sondergaard dissection (separation of the pulmonary veins from the right atrium) is carried out thoroughly (Fig 1) This dissection is continued to separate the right and left components of the atrial septum. The ungloved left forefinger is then inserted into the atrium through the appendage as in the method of Bailey If the defect is operable, sutures are inserted with the use of a two-pointed needle, similar in design but much larger than the type used by Dr R. A. Cowley for blood vessel anastomosis (Fig 2) The needle passes from behind through the posterior rim of the orifice or just to the left of it. It passes through the anterior rim and the point emerges from the atrial wall at any convenient point. The point is grasped with the needle-holder and the direction of the needle is reversed as soon as the trailing point of the needle has cleared the anterior rim of the orifice, and this point is made to emerge posteriorly a millimeter or two from the original point of entrance of the needle. The needle is then withdrawn from the atrium, and this suture, involving only the two rims of the orifice, is tied (Fig 3) Successive sutures are placed until the defect is completely closed. At times, it may be advantageous to pass the needle through the anterior rim after its

direction has been reversed. The small holes in the atrial wall made by the emergence of the point do not bleed significantly.

This "atrial wall conserving" type of atrioseptopexy is advantageous because there is no danger of compromise of vena caval flow from above or below, even in very small children with large defects.

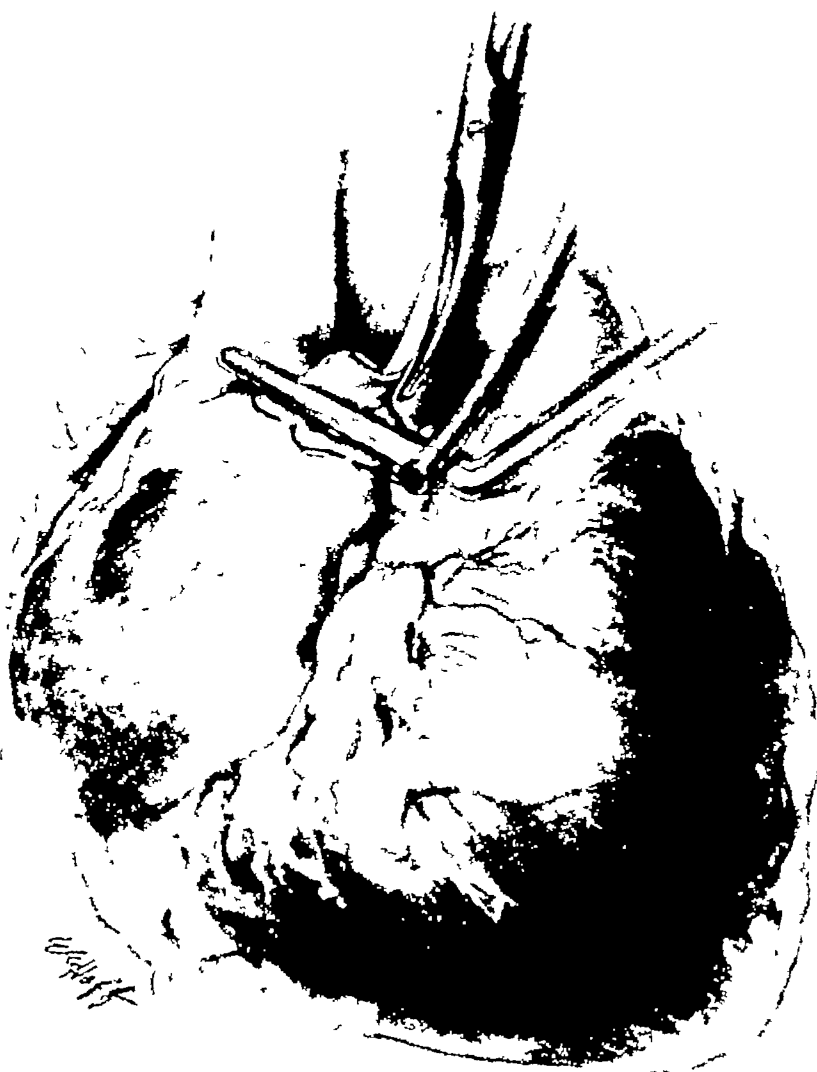


Fig 1. Closure of interatrial septal defect Note the dissection of the pulmonary veins from the right atrium (Sondergaard dissection)

This closed method is not applicable to the ostium primum defects It is my plan for the future that when one of these defects is encountered, I will not waste time trying to close it by a conventional method, but will immediately refrigerate the patient (e g., by the veno-venous method described by Brock), open the atrium and carry out the necessary plastic procedure.

We have carried out some experiments in our laboratory in which the brain has been protected by hypothermia, the heart has been stopped by the injection of 1 cc./kilo of 5 per cent potassium chloride in the left ventricle, the interatrial septum has been replaced by a pedicled flap of right atrial wall, and a pedicle flap of pericardium used to close the resultant defect in the atrial wall

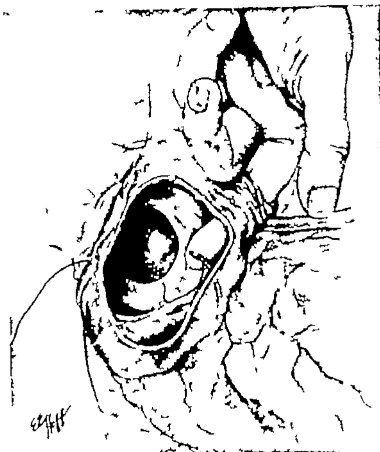


Fig. 2. Placing of first suture to close interatrial defect, using a two-pointed needle with the silk swaged on in the middle. (Ethicon Suture No D–54–78)

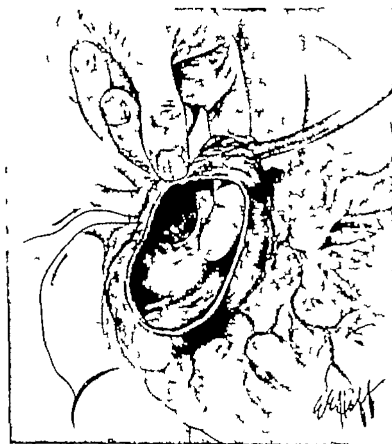


Fig. 3 Two sutures have been tied and third is ready to be tied during closure of atrial septal defect.

DR. SHUMACKER

Dr. Crafoord, would you like to continue this discussion and tell us something about the technique you employ yourself?

DR. CRAFOORD

The technique we use is also based on the dissection of the two atria from each other on the right side between the right pulmonary veins and the vena cava, which Dr. Blalock has shown so beautifully it is possible to perform when he did his cases of transposition, and which have been taken up by Dr. Sondergaard for the first time for the purpose of closing interatrial defects.

We use a circular suture around the defect. The suture enters most often at the root of the aorta. Actually, it goes between the aortic wall and the atrial wall down to the top of the intraventricular septum.

The needle has to be rotated to the left side so that the bundle of His is avoided. Then the needle passes into the muscular substance of the interventricular septum or the base of the interatrial septum.

The needle comes out a little to the left of the base of the interatrial septum behind the vena cava inferior, just above the coronary sinus. The Sondergaard-Blalock groove has already been dissected. The thread is taken up. It comes out behind the superior vena cava, and the suture is tied over a piece of free muscle which is dissected out of the latissimus dorsi muscle.

The graft is then placed into the dissected groove between the two atria. The dissection of the groove can be brought down practically to the brim of the rest of the interatrial septum. It is placed in this groove and then the suture is tied on top of that muscle graft, which approximates the bottom of the groove over to the deep side of the atrial septal defect at the same time as the atrial septal defect is gradually constricted. With a finger in the defect one can feel how the suture gradually closes the defect.

One of the advantages with this method is diminished risk to the bundle of His. I will not say there is no risk, because I stitched into the bundle of His once. I have closed very large interatrial septal defects, also defects of the primum type.

DR. SONDERGAARD

In the last few years several methods for operative treatment of interatrial septal defects have been advocated and tried clinically.

You are all familiar with the splendid work of Bailey and his associates. He has perfected the method of atrioseptopexy, and in patients with an anomalous pulmonary vein entering the right atrium and an atrial septal defect, atrioseptopexy is naturally the procedure of choice.

Gross prefers to operate through a well sutured to the right atrial wall and the method has in his skilled hands proved to be a good procedure.

Lewis and Taufic—and Swan and his group—have used hypothermia and direct suture of the defect under vision after clamping of the inflow tracts of the heart with good results.

These are the most widely accepted methods at present. However, in 1950 our group in Copenhagen first performed the procedure which we now call



Fig. 1 The atrial septum of a dog circumcluded in November 1950 VCI inferior caval vein. VCS superior caval vein. RV right ventricle. R.Au. right auricle. Folds and furrows are seen to radiate from a point just above the tricuspid valve.



Fig. 2. A dog operated on in the spring of 1951 The encircling suture has been placed too far to the right (indicated by the black arrow) and has not been tied sufficiently to close the defect. VCI. inferior caval vein. VCS superior caval vein.

circumclusion of an atrial septal defect on a dog after dissecting the cleavage. The suture encircling the atrial septum was placed blindly—without a finger in the heart—and it was difficult to place the suture correctly.

In another series of dogs with experimentally produced atrial septal defects a semicircular suture was employed with success (Sondergaard et al., 1952), but in patients operated on with this method the defect was not always closed. After a similar experience Björk and Crafoord employed a circular suture but improved the method with a guiding finger in the right atrium.

We went back to the laboratory, and a study of the anatomy of the interatrial septum disclosed that the loose connective tissue and fat in the cleavage

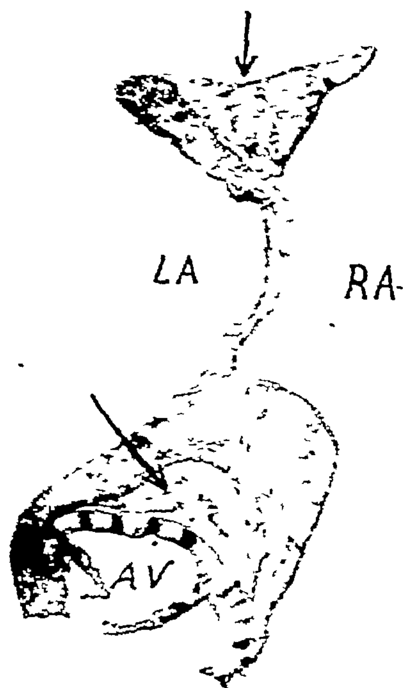


Fig. 3.

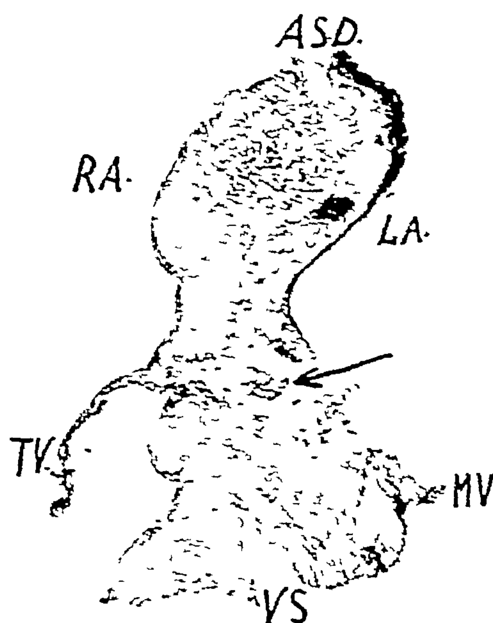


Fig 4.

Fig. 3. A section of the interatrial septum of a dog. LA: left atrium. RA: right atrium. AV. the aortic valves. The oval fossa is in the middle. The two arrows indicate the loose connective tissue The upper arrow points to the cleavage which is developed down to the point where the two atrial walls join to form the septum. The lower arrow points to the track through which the probe is passed.

Fig 4 Section through the lower edge of an atrial septal defect. RA. right atrium. LA left atrium. ASD. the defect. TV: tricuspid valve MV mitral valve VS: ventricular septum The arrow points to the loose connective tissue through which it is possible to pass a blunt probe

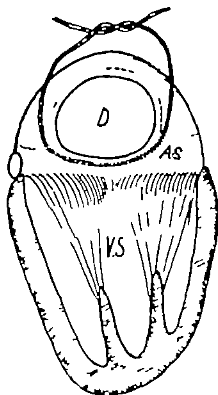
between the muscular walls of the right and the left atrium actually encircles the oval fossa. In other words: the two muscular walls separate again, just over the interventricular septum.

As the dissection of the cleavage between the two atria usually is very easy and mostly blunt, it occurred to us that it ought to be possible to pass a blunt probe along this natural track of loose connective tissue in the interatrial septum, just above the interventricular septum. In the post-mortem room it was very easy to dissect the cleavage and pass the probe. We then

did a series of operations on dogs in which the atrial septum was circumcluded without mortality The dogs were sacrificed some months later and all operations were a complete success In one dog the two atria were completely separated.

We were then ready to try the method in patients In the two years the group has been working in Aarhus, Denmark, we have diagnosed 8 atrial septal defects. Six of these have been operated upon in the last six weeks.

TECHNIQUE OF THE CIRCUMCLUSION OPERATION The right chest is entered through the bed of the sixth rib, and the pericardial cavity is opened



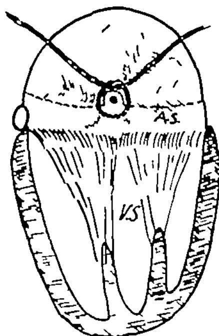


Fig. 5 Fig. 6

Fig. 5 Schematic drawing of the septum after the suture has been placed. The dotted line indicates the developed cleavage. D the defect. AS the atrial septum. VS ventricular septum.

Fig. 6. As Fig. 5, but after the suture has been pulled tight and the defect closed.

extensively 1 cm. in front of the phrenic nerve. The edges of the opening are sutured to the chest wall while care is taken to liberate the caval veins sufficiently from the pericardium, to prevent kinking The two veins are encircled by a heavy suture to facilitate the dissection. The cleavage between the right and the left atrium described by our group in 1952 is now dissected out as far down as it goes Usually a small vessel is cut just in front of the pulmonary veins—otherwise there is no bleeding

The right forefinger is inserted into the atrium through the appendage and the type, size and location of the defect is determined.

An ordinary blunt probe is bent in a suitable curve—the tip is placed at the upper end of the cleavage—and with a very slight pressure the tip disappears into the tissue. Almost by its own weight and by the finger in the

atrium the probe is guided in the tissue along the lower edge of the defect, until the tip appears on the surface of the heart in the lower end of the dissected cleavage in the triangle of fat located between the right and the left atrium and the venous sinus of the heart. It is important that no force be applied to the probe—as long as only slight resistance is encountered the probe is on the right track.

The tip of the probe is caught with a hemostat. An oiled silk suture is tied to the probe, which is pulled back, carrying the suture. The two ends of the suture are now tied over a piece of Gelfoam placed in the bottom of the cleavage. As the suture is pulled tight, gradually the finger in the heart feels the defect becoming smaller and smaller until finally it closes.

No more tension should be used The finger is removed from the heart and the atrium closed. The pericardium is closed partially with a couple of stitches, the pleural cavity is drained and the chest closed in layers.

ADVANTAGES The described procedure is amazingly easy. The nurses and the residents consider a circumclusion of an atrial septal defect and a ligation of a patent duct as operations of the same magnitude. In the last patient operated upon six days ago, the passing of the probe and tying of the suture took less than half a minute.

The advantages of circumclusion can be listed as follows.

1. The established septum is located in the normal septal plane and consists of normal atrial septal tissue
2. Neither the right nor the left atrium is altered in shape or in function, and there is no possibility of blocking of the inflow tracts of the heart.
3 The technique is the same in multiple defects.
4 No sutures or other foreign bodies are left exposed inside the heart.
5 No needle is used and the blunt probe eliminates the danger of piercing the aortic wall—a mishap reported a couple of times in the literature.
6. There is no need for hypothermia or any other elaborate procedures—just ordinary anesthesia as in any other thoracic case.
7. Only standard right thoracotomy is used.
8. The probe is sliding parallel to the described course of the bundle of His—not at a right angle as in direct suture or in the procedure of atrioseptopexy.
9. The mortality has been zero in the experiments and in the clinical cases.
10. All the patients catheterized postoperatively have a complete closure of the defect, and the pressures have returned to normal in two weeks.

DR. SHUMACKER

The next person I am going to call upon is Dr. Kirklin, who is going to mention very briefly the technique he uses, which is Dr. Gross's atrial well method, and make some comments about some postoperative studies of patients.

Before I call upon him, however, I might take one moment to describe an operation my associates and I worked out some years ago, before these simpler and very good methods were available to us. We find it is of value in occa-

sional cases We did not realize it at the time, but it is an amplification of the method that Dr Bailey has utilized, in that one simply increases the size of the atrial wall by suturing a pocket to the edge of the incision. At first we used a pericardial sac (Fig 1), but we now use a pocket made of finely woven

Fig. 1 Drawings illustrating method of closure of atrial septal defect. See text.

nylon. The defect can be easily felt through the invaginated pocket and one of the walls can be sutured to the rim of the defect. One can have a finger in the atrium for guidance. The results in a small number of cases in which it has been utilized have been quite good.

DR. KIRKLIN

In the selection of the proper operative technique for repair of atrial septal defect, it is important to realize that several very good methods of closure are available. Certainly, completely closed techniques, semi-open technique such as the atrial well, and open techniques utilizing hypothermia and inflow stasis have all given good results. An important consideration is that the operative

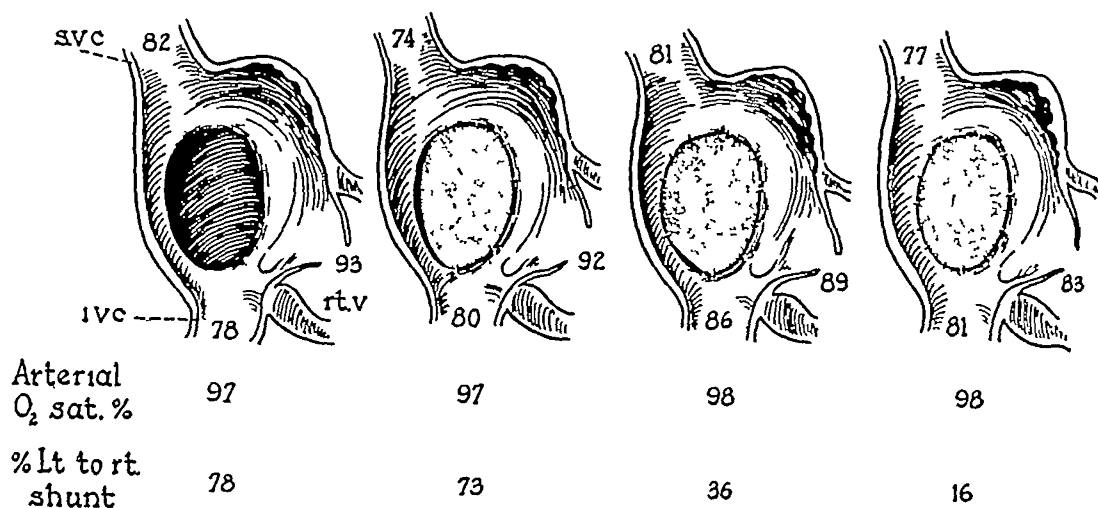


Fig 1. Effect of partial surgical closure on the arteriovenous shunt through an atrial septal defect in a 34-year-old woman At operation through the atrial well, samples of blood for oxygen analysis were withdrawn from the superior vena cava, inferior vena cava and right ventricle These studies were made before, and at varying stages during, closure of the defect With the defect wide open, there was a left to right shunt of 78 per cent With the sponge in place but with the slitlike defect remaining posteriorly, a shunt of 73 per cent persisted. With an even smaller defect remaining posteriorly and inferiorly, there was still a residual shunt of 36 per cent. With a very tiny defect remaining near the inferior vena cava after another stitch, there was a shunt of 16 per cent. Further stitches resulted in complete repair of the defect This emphasizes the need for perfect anatomic repair (Courtesy of Drs W. H Weidman and E H Wood.)

method utilized satisfy three criteria: (1) It must be associated with a very low mortality rate. (2) It must ensure complete and secure closure. (3) It must possess sufficient versatility that anomalous pulmonary venous connections and persistent atrioventricular canals (so-called ostium primum defects) can be satisfactorily managed should they be encountered unexpectedly

In our experience, up to March 1, 1955, with 33 cases of atrial septal defects with predominantly left to right shunts, my colleagues and I have utilized, for the most part, the atrial well of Gross. We have found that this method satisfies the three criteria noted above. Many of these patients have been adults, some with severe pulmonary hypertension, in spite of which there has been only one death in this group of cases. The low operative

mortality of one death in 33 cases seems to lend support to our continuing use of this method. The future, of course, may bring revisions in our ideas

Figure 1 illustrates that very small residual defects may allow significant left to right shunts This emphasizes that whatever technique is decided upon must produce complete anatomic closure.

Finally, our experience with 5 cases of atrial septal defect and anomalous pulmonary venous connection (Fig 2) has shown that the atrial well technique or a modification of the Bailey technique allows handling of this complicated

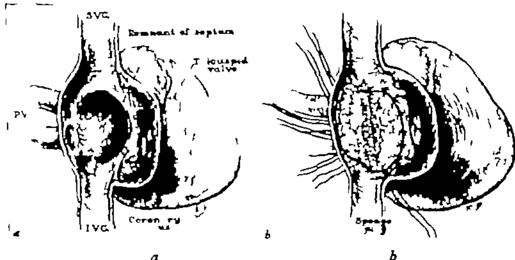


Fig. 2. *a*, Atrial septal defect with anomalous pulmonary venous connection of the right lung to the right atrium. *b*, Repair of this defect through the atrial well The sponge is so placed that blood from the right lung is diverted into the left atrium. (Reproduced with permission from Kirklin, J W, and others. J Thoracic Surg, vol. 29 Jan. 1955)

defect in an exceedingly satisfactory manner Furthermore, in two cases of persistent atrioventricular canal (Fig 3) (so-called ostium primum defects), the openings have been completely closed through the atrial well.

The hemodynamic results of the closure of the atrial septal defects in the first 33 patients operated on by us are presented in Fig 4 Without going into detail, it should be noted that 29 patients have excellent results as judged by physiologic studies either immediately after closure or in the late postoperative period. The two fair results were obtained early in our experience. These two patients had a residual left to right shunt in the neighborhood of 30 to 40 per cent. Both had left to right shunts of 70 per cent preoperatively Although the results in these cases are not excellent, nonetheless they represent significant improvement in the patients condition and certainly these patients would not require reoperation. The one poor result was obtained in the very first patient operated on by us and represents a significant residual defect. The one death was in a 48-year-old woman with severe pulmonary hypertension, associated mitral insufficiency and congestive heart failure. These results demonstrate, as do the results of others, that repair of atrial septal defects may be accomplished at very low risk and with excellent results

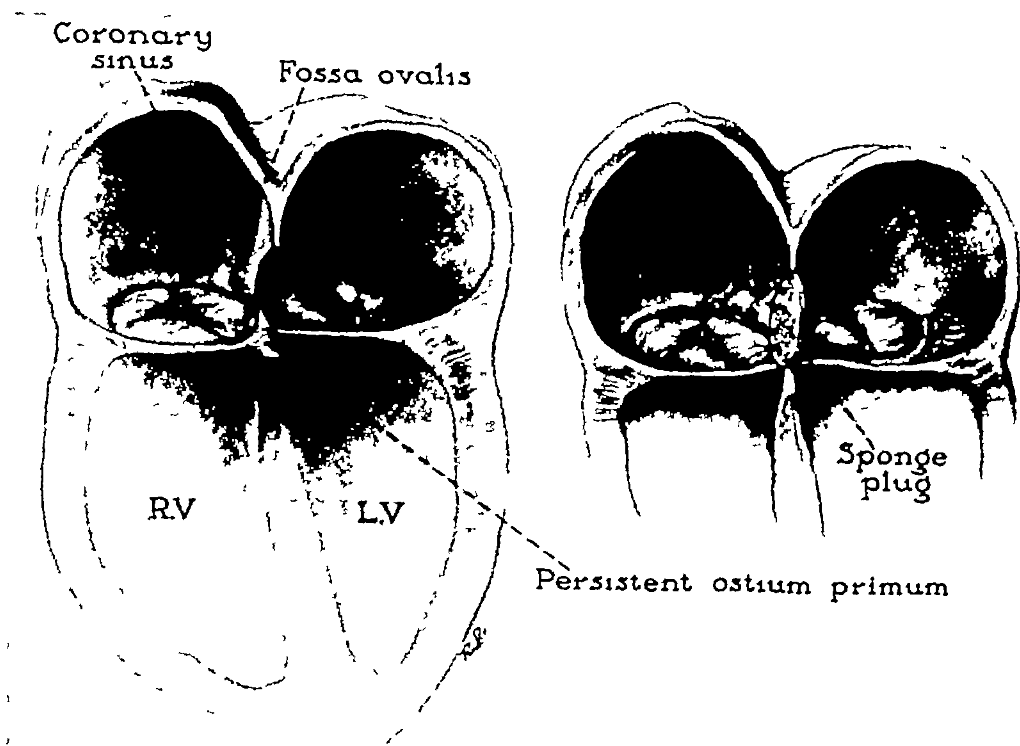


Fig 3 Schematic representation of the technique of repair of persistent atrioventricular canal (so-called ostium primum defect) through the atrial well with a plug of Ivolon sponge. (Reproduced with permission from Kirklin, J W, and others. J. Thoracic Surg, vol. 29, Jan 1955)

HEMODYNAMIC RESULTS OF CLOSURE OF ATRIAL SEPTAL DEFECTS

Defect		Results: Excellent	Fair	Poor	Dead	Total
Atrial septal defect	Without associated defect	19	2	--	--	21
	With anomalous pulmonary venous connection	4	--	1	--	5
	With other associated defects	3	--	--	1	4
Partial persistent common atrioventricular canal		2	--	--	--	2
Common atrium with anomalous membrane (cor triatriatum)		1	--	--	--	1
Total		29	2	1	1	33

Fig 4 Hemodynamic results in the first 33 patients operated on at the Mayo Clinic for atrial septal defect with left-to-right shunt

DR. SHUMACKER

There is one very important technique which has not been mentioned thus far, and that is the method of operating through the widely opened right atrium. At the present time this is being done in a number of clinics under conditions of acutely induced hypothermia.

The major experience, as you know, has been obtained by Dr Swan and his associates in Denver, and Dr Lewis and his associates in Minneapolis Dr Blount, would you be good enough to give us a physician's impression of this technique?

DR. BLOUNT

I am glad you qualified that. Not being a surgeon, some of these other techniques appear quite difficult. However, with the open method, as I tell

TABLE 1 CHANGES IN PULMONARY ARTERY PRESSURE FOLLOWING CLOSURE OF ATRIAL SEPTAL DEFECT

Patient	*Pre-op P. A. Press.*	*Post-op P A Press*	*How Long After*
1 G H.	100/30	30/17	4 mos.
2. E. M.	56/12	24/3	
3 D G	51/35	30/12	2 wks.
4. C. G	49/18	35/11	7 wks.
5 J G	45/5 R.V	44/13	3 wks.
6. W G	38/15	30/18	7 mos.
7 R. C.	30/11	34/17	2 wks.
8. C. C.	27/16	20/10	3 mos.
9 Joyce G	26/4 R.V	15/7	7 days
10. A. C.	20/4	29/13	3 mos.

TABLE 2. CHANGES IN PULMONARY BLOOD FLOW FOLLOWING CLOSURE OF ATRIAL SEPTAL DEFECT

Patient	*Pre-op.*		*Post-op*	
	Systemic Ind. $l/m^1/m^2$	*Pulmonic Ind.* $l/m^1/m^2$	*Systemic Ind.* $l/m^1/m^2$	*Pulmonic Ind.* $l/m^1/m^2$
1 A. C.	6.8	40 0	4.7	4.7
2. C. C.	3.6	13.2	6.1	6 1
3 R. C.	5 0	11.3	5 18	5 18
4. C. G	2.9	6 6	2.3	2.3
5 D G	4 0	13.0	4.9	5.9
6 J G	6.6	15.2	4.5	4.5
7 Joyce G	2.9	9 7	5.3	5.3
8. W G	5 7	13.5	3 8	3 8
9 G H.	2.9	8 9	3 0	3 0
10 E. M.	3.3	7 0	2.9	2.9

Dr Swan, anyone who can sew up a hole in the toe of a sock can close the defect.

We now have closed 24 secundum defects with 2 deaths. Tables 1 and 2 show the evidence of complete closure in the first 10 patients The asterisk in Table 2 shows that there is still some left to right shunt. This patient did have two or three small pulmonary veins. Dr Swan was able to close the

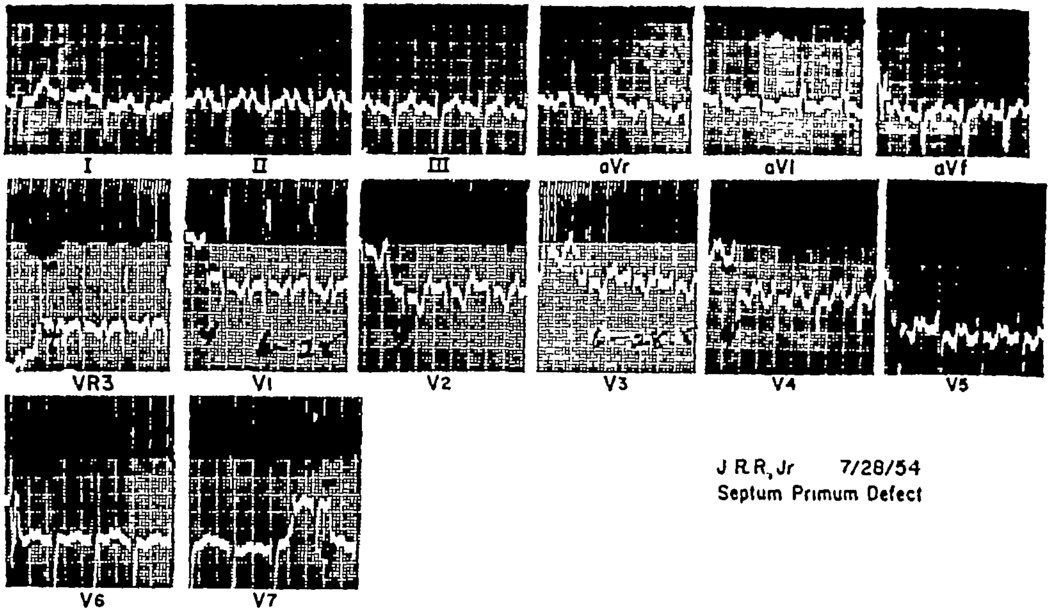


Fig. 1 Electrocardiogram one year after closure of atrial septal defect



Fig. 2 Postmortem specimen showing large defect completely closed See text

defect in such a way that most of the pulmonary venous drainage was returned to the left atrium, but there was one small one remaining.

Figure 1 indicates the changes that occurred in the electrocardiogram one year after surgery In the earlier pictures, we notice the typical picture of partial right bundle branch block, and a year later we note the striking changes in both the limb leads and the precordial positions.

Figure 2 shows a very large defect in a 23-year-old woman, who died on

the fourth postoperative day apparently of a pulmonary embolus There was no clotting over this area, and the defect was completely closed.

Of the 24 who have now been operated on, 19 have been recatheterized, and there is evidence of complete closure in all but 3 In the fourth from the last patient there was a huge defect measuring about 2½ by 3 inches, and in the usual method of placing mattress sutures at intervals the defect was obviously so large that it was not completely closed. Since that time Dr Swan has employed a running suture, anchored on both ends, which seems to close them completely

DISCUSSION

Thomas J E. O'Neill (*Philadelphia*)

Another method useful in closure of atrial septal defects deserves description. It does not require interference with normal blood flow or heart action. The principle is based on an instrument especially devised for intracardiac suturing The instrument can be placed within the heart from any con-

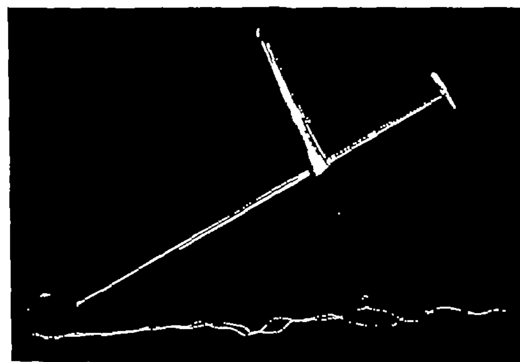


Fig. 1 Intracardiac stitcher The instrument is essentially a curved needle on a hollow shaft—the shaft containing a straight reciprocating needle which will pick up a loop of silk thread carried by the curved needle. In operations, tissue to be sutured is first picked up with the curved needle, after which a straight needle recovers the leading end of the thread. All ends are then brought out so that the knot can be completed.

ceivable angle and can accurately pick up various tissues within the heart and place individual sutures. While this method is especially adaptable to small defects, I have accomplished closure in 7 instances of septum primum defects with no deaths due to operative technique.

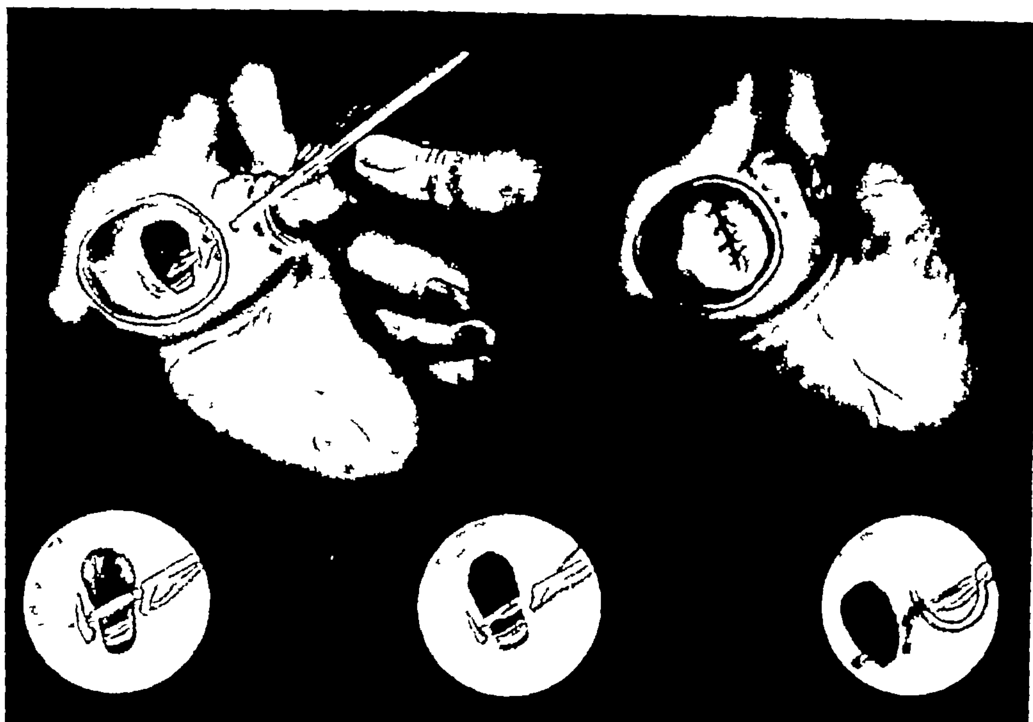


Fig 2 The instrument is demonstrated in action showing the various steps in placement of the sutures The suture ends all come to the outside of the heart when the instrument is withdrawn. A knot is begun on the outside by making hitches on the one hand with the looped end and with the double strand on the other hand, and each hitch is pushed through the small hole left by the instrument until a secure knot is obtained The ends of the sutures are cut flush with the knot allowing the septum to return to normal position.

DIRECT VISION INTRACARDIAC SURGERY

By Means of Controlled Cross Circulation or Continuous Arterial Reservoir Perfusion for Correction of Ventricular Septal Defects, Atrioventricularis Communis, Isolated Infundibular Pulmonic Stenosis and Tetralogy of Fallot

C WALTON LILLEHEI, MORLEY COHEN,* HERBERT E WARDEN,** RAYMOND C READ, RICHARD A. DEWALL, JOSEPH B AUST AND RICHARD L VARCO (*Minneapolis*)

The performance of curative surgery for many cardiac disorders requires a safe and effective method for working within the chambers of the open heart for at least 20 to 30 minutes The attainment of this goal without an unreasonable risk to the patient has required the development of simple methods for totally by-passing the heart and lungs which are sufficiently safe and reliable that the surgeon may with equanimity of mind devote his attention to the many vexing problems associated with the acquisition of a familiarity with intracardiac surgical anatomy and pathology under in vivo conditions, as well as new operative techniques to deal with these lesions

Two methods for open cardiotomy which have appeared in the laboratory to offer workable solutions to these objectives and which have been tested *clinically with success will be described herein.* These two methods are: (1) controlled cross circulation,[1-4] and (2) continuous arterial perfusion from a reservoir of arterialized-venous blood.[5]

GENERAL CONSIDERATIONS

Before describing in some detail the clinical application of these two methods, a discussion of certain general physiologic and technical considerations related to perfusion methods of open intracardiac surgery is pertinent.

SYSTEMIC PERFUSION AT REDUCED RATES OF FLOW The *appreciation of* the low flow concept, because of the simplifications in methods made feasible, has been the single most important factor responsible for the success of these *methods* Andreasen and Watson[6] and Cohen et al.[7 8] found that dogs uni-

* Life Insurance Medical Research Fellow

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formly survived long periods of complete vena caval occlusion at no temperature and recovered without discernible sequelae when only the l flow from their azygos veins was allowed to enter the heart. Cohen measured this azygos vein flow in dogs under these conditions and fou to be 8 to 14 cc./min./kg. of body weight, or slightly less than 10 per of the generally accepted basal canine cardiac output.

Based upon these azygos flow experimental studies, systemic perfusio the patients during the total by-pass interval has been carried out at stantially reduced rates of flow (⅛ to ¼ of the resting cardiac output normal individual of comparable size and weight). These volumes have va from 16 to 40 cc. of blood per kg. of patient body weight per minute in patients having their heart and lungs totally by-passed for periods of 5 to 40 minutes. In these patients there has been not a single instanc cerebral, hepatic or renal dysfunction attributable to these lowered flows

CONTROL OF BODY TEMPERATURE. Patients (particularly infants children) subjected to anesthesia and a bilateral thoracotomy in an air ditioned operating room often undergo a rapid and spontaneous fall in b temperature (3 to 5 degrees per hour). This fall in body temperature h deleterious effect upon the cardiac conduction system and, if severe, preclude successful procedures within the ventricles. As a consequence electric heating blanket is placed under all patients being operated upon intraventricular lesions, and a temperature recording catheter is also inse into the rectum and connected to a dial at the head of the table whe may be monitored by the anesthesiologist to keep the patient's body te perature normal throughout the operation. Moreover, the bank blood transfusion into the patient for loss replacement is warmed to 38° C. i water bath kept in the operating suite. At normal temperatures the myo dium has a significantly greater resistance to conduction abnormalities s as ventricular fibrillation and complete heart block.

BLOOD-FREE INTRACARDIAC OPERATING FIELD. One of the freque voiced handicaps suggested as inherent in a perfusion method for perform intracardiac surgery has been the fact that the intracardiac operating fi would be bathed by the coronary venous circulation. In several repor operations with heart-lung machines utilizing large perfusion flows, this co nary loss has been said to be tremendous. With the techniques that we h used for cardiac by-pass, the patients have been carried at rates of fl substantially below their basal cardiac output. This fact has accounted fo drastic reduction in the quantity of their coronary sinus loss, and has i proved the surgeon's intracardiac visibility immeasurably. However, early our experience it became apparent that blood flowing back into the he through an aortic valve rendered temporarily incompetent by manipulati could be troublesome.

To avoid this annoyance, the root of the ascending aorta has been dissect free from the adjoining pulmonary artery and encircled with a cotton ta

* In addition to the 40 patients herein reported, 11 other patients have had simi defects successfully corrected utilizing a biologic (dog) lung oxygenator, and a simi disposable artificial oxygenator

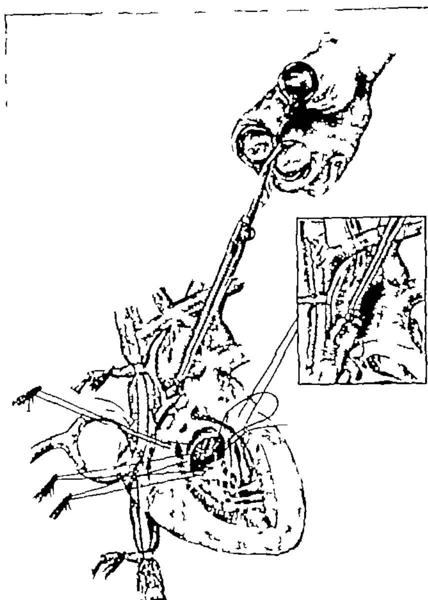


Fig. 1 The aortic tourniquet permitting a bloodless intracardiac operating field The aorta is encircled, as shown routinely in all procedures in which the heart is to be opened. Note in the main drawing the position of the tape temporarily occluding the ascending aorta between the arterial inflow catheter in the right subclavian artery and the aortic valves and coronary artery orifices. This maneuver gives a relatively bloodless intracardiac field at will with no ill effects so long as the time limitations described in the text are adhered to Inset shows the Rumel tourniquet released during the phases of the intracardiac procedure in which a bloodless field is not needed.

to which is attached a Rumel tourniquet (Fig. 1) Then if the cardiotomy field is bloody, the Rumel tourniquet is occluded briefly, while the defect or other intracardiac pathology is inspected, and released momentarily to permit a free coronary flow; and then periodically tightened and opened as required during the actual suturing When needed, the field can be made bloodless, and by this very fact this maneuver has contributed substantially to a reduction in the blood loss via the coronary flow as well as to a reduction in the time needed for the necessary reparative surgery The demonstrated toler-

ance of the heart* for brief episodes of complete stasis of the coronary flow is related to the decreased work load assumed by that organ as a consequence of the total venous inflow stasis. This fact has been emphasized previously in our experimental studies.[7, 8]

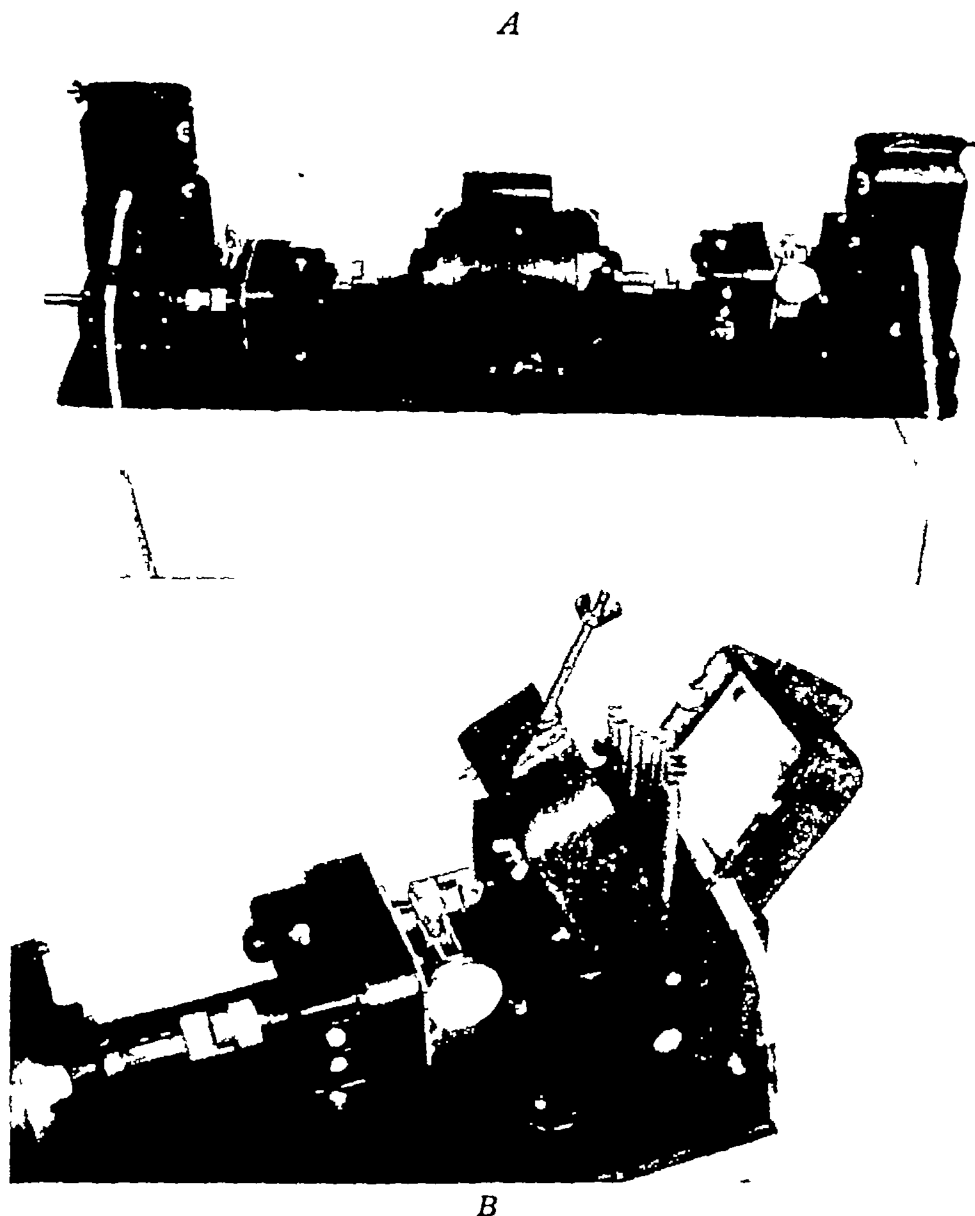


Fig 2 The pump assembly *A*, The pumping unit consisting of a single explosion-proof electric motor (1/3 h p) which activates two pumps each equipped with an individual speed changer and gear box all mounted on a single base *B*, Close-up of one of the pumping units. Note the hinged pump casing for easy loading of the heat-sterilized tubing, the multiple metal fingers, which massage the blood unidirectionally within the tubing, and the speed changer with its vernier control for ease in balancing the flows in the two circuits.

* Dogs with total cardiac by-pass being perfused at reduced flows (25 to 35 cc / kg /min.) from a donor tolerated complete occlusion of their coronary flows for 3 minutes without discernible ill effects. After a 1 minute recovery period, this 3 minute occlusion could again be effected without harm, and the 3 minute occlusion period alternating with a 1 minute recovery interval could be continued more or less indefinitely.

THE PUMP An efficient pump is the only special equipment required for the performance of intracardiac surgery by these techniques.

The pumping unit,* adaptable to either of these techniques, consists of a single explosion-proof electric motor (1/3 h p) which activates two pumps each equipped with an individual speed changer and gear box all mounted on a single base (Fig. 2) The entire unit weighs about 75 pounds The capacity ranges up to a maximum of 2000 cc. per minute through each pump This latter figure represents a considerably greater capacity than would be required for total by-pass in an adult patient. The flows in the two circuits can be quickly balanced by a vernier adjustment of the speed control. The rate of flow is determined by adjustment of the speed changer and the size† of tubing used.

The top of the pump casing is hinged (Fig 2B) to allow easy insertion of the heat-sterilized tubing The two halves of the top open and the tubes are inserted in their proper slots in the side plates The halves are then closed around the tubes and locked in place.

ANESTHESIA. In the cross circulation procedures both the donor and the patient are anesthetized. As a rule various combinations of nitrous oxide or cyclopropane have been employed for the patient's induction and then a change-over made to Pentothal-curare for the maintenance. The donors have usually received intravenous Pentothal-curare as their anesthetic agent.

During the actual by-pass interval the donors have been hyperventilated with 100 per cent oxygen. The value of increasing the donor's respiratory minute volume by hyperventilation with oxygen in order to maintain a near normal pH in both the donor's and the patient's blood during the by-pass interval has been demonstrated in our previous experimental studies [9] By maintaining the donor's alveolar pCO_2 well below the normal range, the effects of the metabolic acidosis which regularly develops in the patient during the total by pass interval are lessened. The patient's lungs during the total by-pass interval are allowed to collapse out of the surgeon's way

Both donor and patient are kept in a relatively light plane of general anesthesia at all times The difference in size and body weight that has existed between the adult donors and many of these patients has not created any special anesthetic problems, inasmuch as the depth of anesthesia is regulated by the blood concentration of the circulating agent and is independent of the body size.

* Sigmamotor Model T-6S, 3 North Main, Middleport, New York.

† The diameter of the short length of tubing (10 cm.) within the pump is an important factor in determining the quantity output per pump cycle. For example, increasing the internal diameter of this 10 cm. length of tubing within the pump from 3/16 to ¼ inch will almost double the output per minute other factors remaining equal. Because of its superior elastic qualities, gum rubber is recommended for this portion of the circuit within the pump which comes in contact with the metal fingers. Plastic tubing of 3/16 or ¼ inch I.D with a wall thickness of 1/16 inch has been utilized in the remainder of the extracorporeal circuits. Alterations in the internal diameter of the tubing of this portion of the circuit have relatively much less effect upon the resistance to flow· therefore, the diameter of the tubing in this portion of the extracorporeal circuit is kept relatively small to reduce the quantity of blood outside the body

In utilizing the method of continuous perfusion from a reservoir of arterial blood, the anesthetic considerations for the patient during the initial phases of the operative procedure are similar to those described above for cross circulation. During the total by-pass interval the patient's lungs are collapsed and the patient is perfused from a reservoir of previously collected arterial blood. During this interval, which has lasted up to 30 minutes, the patients have generally received no additional anesthetic agent. As a result, at the conclusion of the by-pass interval anesthesia is frequently quite light, but this has been considered a desirable state Should additional anesthesia be deemed necessary during the total by-pass interval, it could easily be added to the arterial perfusion blood in the appropriate concentration.

THE METHODS IN MAN

CONTROLLED CROSS CIRCULATION

Figure 3 illustrates diagrammatically the method of linking together the circulatory systems of the patient and donor for the reciprocal exchange of arterial and venous blood, permitting the heart and lungs of the patient to be totally by-passed for sufficient intervals to allow definitive surgery under direct vision.

For the exposure of the heart and great vessels the patient has been placed in a supine position upon the operating table, and the chest is entered through an anterior bilateral thoracotomy incision in the fourth interspace dividing the sternum transversely.

After the pericardium is incised, the caval entrances at the atrium are dissected from their adjacent cardiac tissues to the azygos and hepatic veins respectively. Cotton tapes are then passed around these veins as indicated in Fig 1.

CANNULATIONS IN THE PATIENT The patient's venae cavae are cannulated by individual catheters inserted through separate stab wounds in the right atrium and then joined by a stainless steel Y-cap adaptor. For this insertion, a small area of the atrium is encircled by a 000 silk purse-string stitch. Then, with a stiletto-bladed knife the tented-up atrial wall is incised and the catheter is slipped into its place in the respective cava* (Figs. 1 and 3) The catheters are then secured by tying the previously placed purse-string stitch. Blood loss during these manipulations has been negligible. (The catheter removal is similarly easily carried out, and after occluding a small knuckle of the atrium with a spoon vascular clamp, a suture is applied to this previously made incision.)

A technical point of some value in patients with very large left to right shunts is to refrain from inserting the caval catheters until just before the

* The catheters utilized to cannulate these vessels in the patients and donors were standard commercially manufactured (Bardic or Pharmaseal) thin walled plastic nasal oxygen catheters 12 f to 14 f in size The 12 f size was utilized in the infants and the 14 f size was found suitable for the older children

We have usually stiffened the catheters for this cannulation by temporarily inserting a No 26 steel wire into their lumen This wire is then withdrawn as soon as the catheter is in place

cardiotomy Such patients frequently have very small cavae (because of their low systemic blood flow), and even the small catheters used may occupy 50 per cent or more of the caval lumen and depress the blood pressure by interference with cardiac filling The same considerations apply to their removal promptly after completion of the cardiotomy and the resumption of a satisfactory heart action. The blood pressure often responds by a further 20 to 40 mm. Hg rise as soon as these caval catheters are removed.

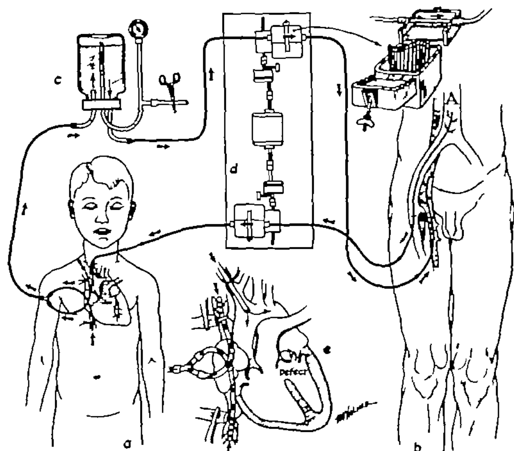


Fig. 3 Method of linking donor and patient for direct vision intracardiac surgery by means of controlled cross circulation. *a*, Patient showing sites of arterial and venous cannulations *b*, Donor, showing sites of arterial (superficial femoral) and venous (saphena magna) cannulations. *c* The venous reservoir and air-trap *d* The pump assembly Consists of a single electric motor and two pumping units each with its own speed changer This pump controls the reciprocal exchange of blood between patient and donor *e*, A magnified view of the patient's heart showing the two plastic vena caval catheters inserted through separate stab wounds in the right atrium and positioned with their tips in the superior and inferior vena cava respectively, so as to aspirate venous blood from both caval systems during the by pass interval. Note also the relative position of the vena caval occluding tapes for securing inflow stasis during the intracardiac procedure. The arterial blood from the donor is circulated to the patient's body via the catheter inserted into the right subclavian artery

The patient's arterial catheter has been inserted into either the right or left subclavian artery and positioned so that its tip lies in the ascending aorta. The introduction of this catheter has been facilitated by mobilizing the artery by ligation and division of its branches followed by transection of the main subclavian artery at the apex of the chest. Following decannulation the proximal artery has been ligated.

DONOR CANNULATIONS. The donor cannulations have been performed through a short incision two fingerbreadths below either the right or left inguinal ligament exposing the great saphenous vein and the superficial femoral artery. The donor's venous catheter has been threaded into the saphenous vein so that its tip lies in the iliac vein or inferior vena cava. The donor's arterial catheter has been inserted into the superficial femoral artery through a small incision and is positioned so that its multiple-holed tip lies within the abdominal aorta (Fig. 3*b*). This latter maneuver is vital to assuring an adequate supply of blood at all times and has eliminated the possibility of the vessel wall collapsing about the catheter and thus obstructing it during the perfusion (as might be possible were this catheter within a smaller branch of the aorta).

ANTICOAGULANTS. Just prior to the insertion of these catheters, both the donor and the patient were each given a single intravenous dose of heparin (0.75 mg. per pound body weight). Following conclusion of the intracardiac procedure the patient usually has been given a similar quantity of protamine sulfate, although in a number of patients the use of protamine has been omitted with no detectable ill effects. No protamine has been given to any of the donors. These relatively small doses of heparin suffice in the set-up because of the limited surface area of the extracorporeal contacts for the blood. In addition, abnormal postoperative hemorrhage has not been a problem, quite likely because platelets and fibrinogen are not removed from the blood in this system by contact with relatively large areas of foreign surface.

VENOUS RESERVOIR AND BUBBLE TRAP. The venous reservoir (Fig 3*c*) serves two important functions, each of which augments the safety of the controlled cross circulation method. The first of these functions is to maintain a free flow of blood out of the patient's caval system under all conditions by preventing the development of an elevated negative pressure in the afferent venous circuit, which tends to occlude the catheter tips by causing the thin caval walls to be sucked into the catheter ostia. The second important function of the reservoir is to serve as an effective bubble trap This function is especially valuable when it becomes necessary to work within the right atrium, because it makes it unnecessary to pull the occluding tapes about the cavae so tight that there is risk of obstructing the flow through the contained catheters. Instead these tapes are pulled up very gently, and if any air leaks into the system it is effectively trapped within the reservoir.

This reservoir unit consists of a 1 quart (960 cc) wide mouth glass Mason jar onto which a one-piece stainless steel top has been fashioned. This cover contains three metal cannulas (3/16 or ¼ inch I.D) as illustrated (Fig 3*c*) A regular Mason jar cap and rubber gasket are utilized to affix this new top in place. These three cannulas constitute, respectively, the reservoir inflow orifice, the outflow orifice, and the air inlet which projects above the level of the contained blood when the bottle is suspended in its top down position. The air inlet tube is ordinarily occluded on the outside by a clamp and has at its top a vacuum gauge.* The entire assembly is sterilized by auto-

* This gauge is not essential and has frequently been omitted in more recent perfusions.

claving (except the gauge which needs no sterilization).

After the donor's and patient's vessels have been cannulated and the pump given a final calibration with warm sterile isotonic dextrose solution, the reservoir which has been previously filled with filtered compatible citrated bank blood* is inserted into the extracorporeal circuit by transecting the plastic tubing (which has been left filled with the isotonic dextrose) at the desired position in the afferent venous circuit (Fig 3c) The reservoir is then suspended at a level below that of the patient to provide a gentle siphoning effect upon the cavae and also to prevent any tendency of the pump to draw blood preferentially from the reservoir

After the pump has been started and the cavae occluded, the reservoir is monitored full-time by an assistant. In about two thirds of the cases in which the reservoir has been utilized it has required no further attention during the run. However, in the remaining cases, whenever there was more than the usual slightly negative pressure (−4 to −6 mm. Hg) noted on the gauge, a change also easily detectable by a slight fall in the level of the blood within the reservoir, the assistant monitoring the reservoir has opened the air inlet for 15 to 30 seconds while 30 to 50 cc. of blood leaves the reservoir and the pressures within are readjusted. The air inlet is then generally reclamped (but it may be left open since the pump will not draw preferentially from the reservoir if the flow out of the cavae is unimpeded), and the flow from the cavae proceeds smoothly and freely again for a variable period. It has not been necessary to leave the reservoir open in any case for more than 30 seconds to 1 minute at a time, for this pressure readjustment occurs promptly The fact that a transient release is all that is necessary strongly suggests the absence of any significant failure of venous return† to the heart, but rather indicts obstruction caused by collapse of the vena caval walls about the catheter tips The Sigmamotor pump is so constructed that the tubing is always completely shut off by one or more of the metal fingers, thus once a negative pressure develops within this closed system it is maintained and accumulates unless a method such as the reservoir is provided to correct the situation. In none of the clinical cases to date has it been necessary to release more than a total of 350 cc. of blood from the reservoir during the entire by pass interval.

More recently we have confirmed the suggestion that if the diameter of the

* There is no reason that the reservoir needs to be primed with blood except that the resultant hemodilution in the donor (if it were filled with isotonic glucose or saline) would reduce somewhat the oxygen-carrying capacity of the donor's blood being circulated back to the patient.

† Andreasen and Watson[10] experienced difficulties in getting the blood to flow freely in their caval catheters, and they concluded that significant capillary pooling engendered by low perfusion rates was occurring in their animals. We have not observed this phenomenon nor have we been able to duplicate it in our dogs even at extremely low flow rates for prolonged periods up to 1½ hours. Therefore, we believe that the explanation of these difficulties in related to their method of cannulation of the cavae and possibly also to their pump, and that significant capillary pooling with failure of venous return does not occur In fact, actual pressure measurements confirm the latter by demonstrating the sustained high venous pressures in the cavae outside of the heart during the perfusion interval.[9]

rubber tubing within the pump is increased sufficiently so that the flow is pulsatile with a long diastolic phase (up to 50 per cent or more of the cycle), then this tendency towards a negative pressure build-up is lessened or even obviated as measured by the gauge and reservoir. However, we feel that the other functions of the reservoir are of sufficient importance that it should remain in the circuit. Particularly is this so since the reservoir has proved free of any complications.

We have further investigated the possibility that the flow of blood into and out of this reservoir during the perfusion might cause hemolysis. Measurements of the plasma hemoglobin levels in the reservoir blood immediately before and after perfusion show no increase Likewise, measurements of the plasma hemoglobin levels in the donor's blood immediately following conclusion of the perfusion procedures have remained normal.

In the arterial limb of the extracorporeal circuit, flow is always free so long as the tip of the afferent catheter lies within the donor's abdominal aorta (Fig. 3*b*).

BLOOD LOSS REPLACEMENT. The donor does not experience any net loss of blood during perfusion since the pump is set to return to him the same amount of venous blood as leaves via his arterial catheter. The patient's blood losses from the operative wound and from the blood aspirated from the interior of the heart during cardiotomy are replaced synchronously volume for volume with citrated bank blood introduced via a cannula in the saphenous vein. In none of these patients has the coronary sinus blood aspirated from the interior of the heart been returned to the patient's or donor's circulation because of the trauma (hemolysis) imposed upon this blood by an effective sucker

SELECTION OF DONORS None of the patients or donors undergoing the cross circulation procedure has exhibited any evidence of blood incompatibility reactions. The complete freedom from this complication may warrant an expression of the principles utilized

Good physical health is required, together with freedom from a history of syphilis or a past history of hepatitis or malaria within five years. In addition we have customarily performed a chest roentgenogram, electrocardiogram, complete blood count and a urinalysis on all prospective donors to detect any evidences of serious abnormalities More specifically, the following four general principles are believed to be of value in regard to the blood typing and cross matching.

1. The blood of donor and patient must be homologous with respect to the ABO system. Donors who have not had previous transfusions are preferred because of the increased hazards of possible sensitizations from prior transfusions.

2 The better known blood cell types have been determined (Table 1), and we believe it desirable that they be identical in donor and patient as well as in the other blood to be used for transfusion replacement to avoid potential immunizations The number of such types that have been determined has to a certain extent depended upon the availability of specific antiserums *

* Kell and Cellano antiserums have been supplied through the courtesy of Dr. Philip Levine and Mr Glen Hill of Ortho Research Foundation, Raritan, N J

TABLE 1 SELECTION OF DONORS FOR CROSS CIRCULATION

I. Good Physical Condition and Freedom from *Infectious Disease.*

II Blood Cell Types Determined in Patient, Donor, and Each Bottle of Transfused Bank Blood.

(1) A B O
(2) RH—hr
C (rh')
D (Rho)
E (rh")
c (hr')
e (hr")
(3) M, N
(4) Kell (K)
(5) Cellano (k)
(6) Duffy (fy^a)

III Cross Matching is Performed Between Patient Donor and Each Bottle of Bank Blood for Transfusion by Both
(1) Saline Centrifugal, and
(2) Indirect Coombs Methods

3 If the blood cell types are not identical in the donor and the patient, then potentially harmful immunizations should be avoided for both donor and patient, but particularly the healthy donor deserves this protection against future hazards should further blood transfusions become necessary at a later date. Likewise, in the case of donor women in the childbearing age, future pregnancies might provoke reactions should harmful immunizations be permitted.

4 In the case of potentially less harmful sensitizations, we prefer to have the patient rather than the donor accept the disadvantages of such possible immunizations

When a suitable donor has been tentatively selected in accordance with these criteria, both a saline centrifugal tube cross match and an indirect Coombs' test are performed between patient and donor Similarly, the serum of the patient and donor and cells from each additional bottle of bank blood intended for the patient's loss replacement during the surgical operation are tested for compatibility by the indirect Coombs' test. The serum of each bottle of bank blood is also tested with a saline suspension of cells of both patient and donor The techniques for these tests have been described elsewhere.[4]

CONTINUOUS ARTERIAL PERFUSION UTILIZING ARTERIALIZED-VENOUS BLOOD

The arrangement of the extracorporeal circuits for this method is diagrammatically portrayed in Fig 4 It may be noted that the general plan is similar to that employed in the controlled cross circulation method described above with the exception that the donor is replaced by arterial (A 1 and 2) and venous (V 2 and 3) reservoirs. These are standard siliconized blood collection bottles (500 cc. or 1000 cc.) from which the vacuum has been exhausted.

Bottles A 1 and 2 contain arterial blood that has been previously collected in the manner to be described below, and V 2 and 3 are empty at the outset and are provided to collect the venous blood* withdrawn from the patient during the perfusion.

The extracorporeal circuits are assembled by first attaching two bottles of arterial blood to an ordinary intravenous blood filter (A 3) and allowing blood

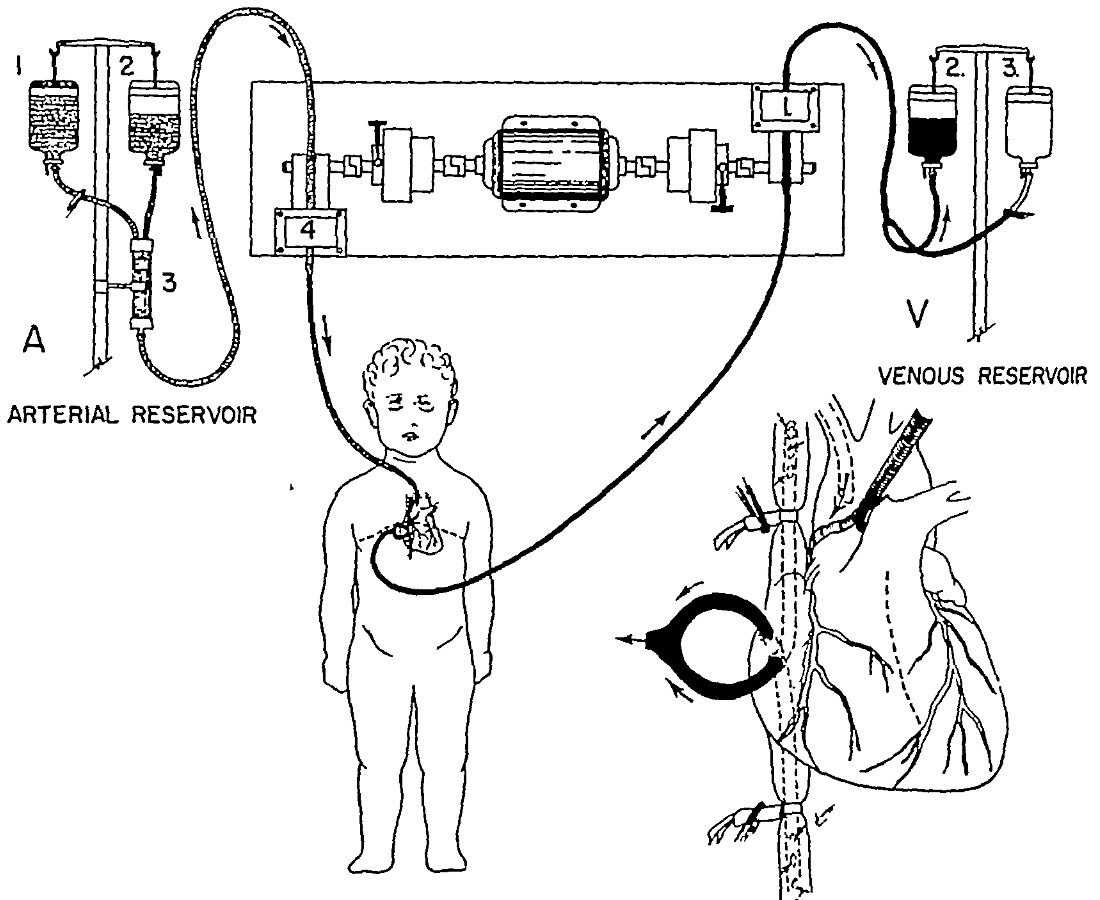


Fig 4. Arterial reservoir perfusion *A*, The arterial reservoir showing the bottles of arterial blood (1, 2), blood filter (3), and arterial pump (4) *V*, The venous portion of the perfusion circuit with the venous pump (1) and the empty bottles (2, 3) for the collection of the venous blood withdrawn from the patient's caval system At the top center is the pumping assembly. The patient's relationship to the system is depicted in the center of the diagram In the lower right is a close-up view of the cannulations about the patient's heart Note A Rumel tourniquet has been placed about the base of the aorta in order to insure a dry intracardiac field

to fill the filter chamber while the latter is held in the inverted position so that all air will be displaced

The outflow from the filter is then attached to the afferent plastic tube of the arterial pump (A 4). The pump is turned on momentarily so that the entire arterial circuit is filled with blood When this is done and the venous circuit is assembled as diagramed, the appropriate connections may be made to the patient's cannulas and the perfusion begun. As illustrated, the arterial

* This blood is utilized to transfuse the patient during or after the surgical procedure as needed to replace losses.

pump draws blood from only one bottle at a time. When the initial bottle is nearly empty, as for example A 2 in the diagram, its outflow is clamped, that of A 1 released, and the perfusion goes on uninterrupted while A 2 is replaced by a full bottle of arterial blood. This procedure is repeated until the pump is stopped. Similarly on the venous side when V 2 is full, the venous outflow from the pump is diverted into V 3 while V 2 is replaced by another empty bottle.

The output of each pump is preset at equal rates before the pump is brought to the operating room. It is desirable to check this calibration just prior to the onset of perfusion. This is done under sterile conditions using a 5 per cent glucose solution in order to avoid any wastage of blood. If desired,

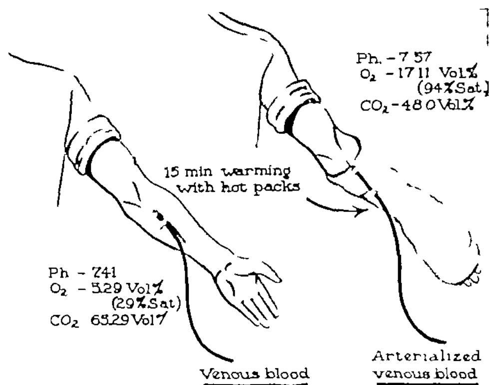


Fig. 5 Method of arterialization of venous blood by cutaneous heat. The results are illustrated for a representative study in which blood samples were drawn simultaneously from the normothermic and the warmed arms of the same individual.

to reduce the quantity of blood necessary for replacement of the patient's losses during the open cardiotomy, the venous outflow pump may be set at a lower rate of flow than the arterial inflow pump

COLLECTION OF ARTERIAL BLOOD This method of reservoir perfusion requires the procurement, collection and storage of arterial blood with which to supply the arterial reservoir Our previous investigations bearing upon these factors are detailed elsewhere,[5] and only those considerations pertinent to the clinical use of this method will be repeated here.

ANTICOAGULANTS. Heparin has been used as the anticoagulant instead of the customary acid-citrate-dextrose (ACD) solution because of the marked reduction in blood pH caused by the citric acid and its toxic effect upon the

myocardium, which is due to the acute removal of calcium ions resultin prompt cardiac arrest. Likewise, the banked venous blood for use in patient to replace the blood lost at operation should be heparinized rat than citrated blood for the same reasons.

The optimum amount of heparin to prevent in vitro clotting without dan of abnormal hemorrhage in the patient was found to be 15 to 20 mg. of hep per 500 cc. of blood (either venous or arterial). In order to enhance mix this amount of heparin was added to 50 cc. of 5 per cent glucose solution the mixture was instilled into each 500 cc. siliconized bottle before collect of the blood. The blood has been collected by gravity to insure maxim platelet preservation and with continuous gentle agitation to prevent formation.

Ordinarily the arterial blood for perfusions has been collected within 2 4 hours of the time it was to be used. During this intervening period it been kept in a waterbath at 38° C. to obviate the previously mentioned dele rious effects upon the heart of perfusion with cold blood.

METHOD FOR COLLECTION OF ARTERIALIZED-VENOUS BLOOD FROM MULTI DONORS. An important contribution to the simplicity and convenience collection of arterial blood for these procedures has been the use of arteri ized-venous blood.

Meakins and Davies (1919)[11] observed that blood samples from the an cubital vein of an arm that had previously been warmed in a waterbath at 45 to 47° C. for 15 minutes were strikingly similar to arterial blood. In order to investigate further the effects of local heat, venous blood samples were drawn simultaneously from the normothermic and the warmed arms of the same person in a series of individuals in our laboratory. A representative study in this series is portrayed in Fig 5.

The mechanism by which this arterialization of venous blood occurs is the result of a local increase in rate of circulation and the opening up of multiple small arteriovenous anastomoses in response to the increased temperature of the extremity. Further, the practical value of these previous physiologic observations was increased when through these experiments it became evident that by continued heating of the arm the resultant arterial character of this blood drawn from the veins could be maintained for any desired quantity

As a result, a simple method of obtaining large volumes of arterialized blood from human donors for clinical use was evolved as follows: The arm and forearm of the prospective donor is immersed in a waterbath at 45° C for 15 to 20 minutes. For the actual collection the donor then reclines on a cot and the waterbath is replaced by hot towel packs around the forearm to maintain the local hyperemia during the collection interval. Then by means of a venipuncture in the antecubital fossa, 500 cc. of arterialized-venous blood is collected by gravity from each of the donors. The collections are carried out in the blood bank by the regular technicians delegated for blood collection duties.

The total volume of blood collected in preparation for a given operation has been determined by the flow rate desired, the weight of the patient, and the estimated length of perfusion needed.

RESULTS

The primary scope of this presentation is to present the technical details of two practical methods for direct vision intracardiac surgery in man. However, the results obtained from the application of these methods to clinical problems of heart disease previously not amenable to intracardiac curative techniques is obviously pertinent and will be briefly reviewed here.

CROSS CIRCULATION The results of the first 39 cross circulation procedures, our experience to date with this method for the defects indicated, are listed in Table 2.

TABLE 2 RESULTS OF DIRECT VISION INTRACARDIAC CORRECTION OF CONGENITAL ANOMALIES BY CONTROLLED CROSS CIRCULATION

Defect	*Direct Vision Procedure*	*Results* No Patients Operated Upon	Survived
Interventricular septal defect	Suture closure of septal defect	25	18*
Tetralogy of Fallot	Suture closure of ventricular defect with resection of infundibular stenosis	10	6†
Atrioventricularis communis	Suture closure of both atrial and ventricular defects with correction of valvular deformity	3	1‡
Isolated infundibular pulmonic stenosis	Resection of infundibular pulmonic muscle	1	1§

* Six patients have now been recatheterized 4 to 6 months postoperatively and in all their prior existing shunt is completely closed.

† The femoral artery oxygen saturations in these patients preoperatively varied from 25 to 80%, postoperatively all are normal (above 94%). Among the 6 successful cases is a patient with pentalogy of Fallot and a patient with atresia of the main pulmonary artery

‡ Has been recatheterized and no shunt remains. The preoperative pulmonary artery pressure has fallen from a high of 90/50 mm. Hg to a postoperative value of 20/4 mm. Hg.

§ Has been recatheterized and the right ventricular pressure, which was 136 mm. Hg preoperatively was (4 mo postoperatively) 25/0 mm. Hg.

There has been no donor mortality in these 39 operations.

Ventricular Septal Defects Our initial and largest experience to date has been with this lesion. All of the 25 patients selected for corrective surgery were clinically judged to be in serious jeopardy and all had pulmonary hypertension. The detailed preoperative cardiac catheterization data and the other evidences of their serious condition have been discussed elsewhere.[1-4] Sixteen of these patients were infants under the age of 2 years

All of these defects have been closed by direct suture (Figs 1 and 6). It is

particularly gratifying that of the 18 survivors, 6 of the first patients have now been recatheterized 4 to 6 months postoperatively, and in all the shunt has been completely corrected. Only one patient developed ventricular fibrillation during closure, and this patient was successfully defibrillated. Of the 7 deaths, none occurred in the operating room, but rather the patients died in the postoperative period. These deaths were due to technical errors (such as imperfect closure of the defect), acute right heart failure due to severe



Fig 6 Closure of ventricular septal defect Photograph taken at operation in a 6 month old infant (Case L O, U. H #880964, see case report in text). Note the cardiotomy incision in the right ventricle with the enlarged pulmonary artery visible above the apex of this incision. The flexible shank of the Rumel tourniquet encircling the base of the aorta immediately adjacent may be seen. At the lower margin of the cardiotomy may be noted the curved aspirator within the right ventricle Immediately adjacent is a curved Rankin forceps grasping the lower margin of the ventricular defect while a silk stitch is being placed there Another closure stitch, already placed and drawn taut, is visible at the apex of the ventricular defect

pulmonary arteriolar intimal proliferation resulting in an intolerable pulmonary resistance (3 cases), or to respiratory complications to which these patients with pulmonary hypertension are particularly prone Six of these 7 deaths were in the infant group, but 10 of these 16 desperately ill infants survived corrective surgery. There were no deaths due to heart block, ventricular fibrillation or hemorrhage.

Tetralogy of Fallot. The next largest group subjected to direct vision intracardiac curative procedures has been those with Fallot's tetralogy In this group, 10 patients, all consecutively operated upon, have had their ventricular septal defects closed by direct suture and their pulmonic obstructions

(usually infundibular) corrected, with 4 deaths All of the 6 survivors have normal femoral artery oxygen saturations and are asymptomatic postoperatively The first 2 patients operated upon have now had right heart recatheterization six to nine months postoperatively The first patient remains asymptomatic but has a small left to right shunt high in the right ventricle. The other boy has completely normal oxygen contents and pressures throughout.

Also numbered among the cases successfully managed by these curative techniques are a 22 month old girl with complete atresia of the main pulmonary artery, and a 10 year old boy with pentalogy of Fallot in which simultaneous atrial and ventricular cardiotomies were performed for correction of his defects A more detailed consideration of the technique of correction utilized in these patients has been presented in another publication.[12]

As a result of these gratifying experiences the curative operation has been adopted in our clinic *as the method of choice for all patients with the tetralogy* of Fallot defect who are in need of surgery at this time. For lesser degrees of disability we recommend no surgical operation at this time, but rather periodic reevaluation in the clinic.

Atrioventricularis Communis This is a complex anatomic defect usually causing clinical difficulty early in life owing to the rapid development of pulmonary hypertension. The operative repair is best undertaken through a right atrial cardiotomy The technique of repair has been described elsewhere in greater detail.[2,3] It should be emphasized that this defect is anatomically a completely curable one in many instances provided open techniques are utilized.

Isolated Infundibular Pulmonic Stenosis One patient with this defect, a 5 year old girl admitted to the hospital with subacute bacterial endocarditis, was successfully managed surgically after her blood stream infection had been controlled by antibiotic treatment.

Donor Results In these 39 operations utilizing cross circulation there has been no donor mortality One serious donor complication occurred. During the by pass interval a mother developed profound hypotension necessitating a thoracotomy with cardiac massage. She recovered without sequelae and was discharged from the hospital in good condition 8 days later This complication was due to a severe disproportion in the reciprocal exchange of blood between donor and patient during the interval of the cardiac by-pass This complication has been obviated in subsequent cases by the routine use of the reservoir (Fig 3*c*) between the patient and the pump in the venous circuit. This reservoir prevents the patient's venae cavae from being sucked up against the catheters and thus obstructing the flow of venous blood from the patient to the donor as occurred in the above-mentioned instance.

PERFUSION FROM AN ARTERIAL RESERVOIR. Prior to the fruition of the above-mentioned investigations upon the effects of cutaneous heat for arterializing venous blood, we had obtained the blood for use with this technique by arterial puncture in multiple donors or from a single donor utilizing synchronous transfusion with venous blood while withdrawing arterial blood.

The first patient to have a ventricular septal defect repaired utilizing the

method of continuous arterial perfusion from a reservoir of arterialized-venous blood is described briefly below to illustrate this method.

Case Report L O, U H #880964, a 6 month old male infant, entered the hospital because of failure to gain weight, dyspnea, a heart murmur heard on physical examination, and frequent respiratory infections during the interval between 3 and 6 months of age On admission the liver was enlarged and cardiomegaly with increased pulmonary vascular markings was noted upon x-ray examination of the chest Cardiac catheterization revealed a jump in the blood oxygen saturation of 24 per cent between the right atrium (62%) and the right ventricle (86%) and a pressure of 45/0 mm Hg in the outflow tract of the right ventricle These findings indicated an interventricular septal defect

The patient was operated upon and a high interventricular septal defect approximately 1 cm. in diameter was found and closed under direct vision by using the multiple interrupted silk suture technique Figure 6 is a photograph taken during the open cardiotomy in this patient showing one of the silk sutures being placed for closure of his ventricular defect During the 14 minutes and 45 seconds of total cardiac by-pass necessary to close this defect, the patient (wt 4 8 kg) was perfused at a rate of 150 cc per minute (31 cc /kg /min) by means of the described reservoir method Arterialized venous blood for the arterial reservoir had been drawn a few hours preoperatively in the blood bank from 8 compatible donors whose arms had been heated to 45° for 15 minutes prior to collection A total of 3950 cc. was drawn in this fashion and available for perfusion, however only 2225 cc was needed. His immediate postoperative laboratory studies revealed a platelet count of 189,000 and normal bleeding and clotting times with a hemoglobin of 13 4 gm. The loss from the chest catheters yielded only 110 cc of serosanguineous fluid during the first 48 hours Postoperatively, the patient's convalescence was uneventful. The cardiac murmur and thrill have disappeared

DISCUSSION

For the seriously ill patient the technique of controlled cross circulation has proved to be physiologically superb. Once the circulatory systems of the patient and the donor have been linked together, the patient has been assured of a continuous supply of oxygenated and otherwise perfectly adjusted blood from his donor. In fact, both in theory and upon the basis of actual clinical experience it might be suggested as unlikely that a technique for total cardiac and pulmonary by-pass will be developed that for the patient's safety excels this one. This conclusion receives added emphasis from the fact that the last 14 consecutive cross circulation curative operations, all carried out in seriously ill infants and children with pure ventricular septal defects or tetralogy of Fallot defects, have been performed with only one death (a 3 month old infant with a ventricular defect) However, in utilizing cross circulation one must be prepared to accept responsibility for the well-being of the normal healthy donor.

This consideration together with the observations made during the use of controlled cross circulation in the operating room, namely, that in infants and small children the total amount of blood exchanged during the perfusion period was relatively small and could be provided by previously collected arterial blood instead of a donor circulation with virtually all of the advantages of the latter technique except unlimited time, led to the development of the reservoir perfusion method Moreover, experience indicated that for

many intracardiac lesions the interval of direct vision required was nearly always less than 30 minutes and usually less than 20 minutes, so that the above-mentioned limitation of time has not been a problem in utilizing this technique in selected clinical cases This is particularly true in regard to application of the method to infants and small children who comprise many of the patients urgently in need of intracardiac procedures In such individuals, as in the case reported, the amount of blood used for the entire perfusion is less than that required to prime many of the existing extracorporeal perfusion systems

The feasibility of this method is further enhanced by utilizing the physiologic phenomenon of the arterialization of venous blood by local heat during the collection of the reservoir blood. In this fashion, the necessary volumes of blood with the chemical characteristics of arterial blood can be drawn conveniently in the blood bank by the usual phlebotomy technique. Arterial blood obtained in this fashion and delivered by the reservoir system provides the patient with the same physiologic benefits of blood adjusted by the homeostatic mechanisms of a normal donor as in controlled cross circulation, but without any possible risk to the donor

The relative freedom in these patients from such expected serious conduction system disturbances as ventricular fibrillation or persistent complete heart block, even though all defects have been closed by direct sutures placed in the ventricular septum with no effort made to avoid the 'so-called" vulnerable areas, has been gratifying. This fact verifies for man our previous experimental observations indicating that the heart at normal temperature and totally by-passed to reduce its workload needs only a greatly reduced coronary arterial flow to remain well oxygenated. Further, even a sick human heart under such conditions becomes resistant to the induction of arrhythmias

In conclusion, upon the occasion of this symposium it is tempting to speculate upon future progress in this dynamic field. The demonstration that patients with interventricular septal defects, atrioventricularis communis, isolated infundibular stenosis and tetralogy of Fallot have had their congenital heart defects completely corrected by open cardiotomy likely will stimulate rapid improvements in these methods as well as the development of other new techniques for total cardiac by-pass As a consequence it is quite possible, even at the present time, to envision corrective surgery for the vast majority of all types of congenital heart defects Moreover, the feasibility and practicability of the direct vision approach to intracardiac lesions of the acquired type is obvious, and likely will make surgical management possible for certain conditions of this type now quite hopeless

The scope of future expansion both necessary and likely in this field is impressive. The fact that more than 50,000 infants[13] are born in the United States each year with congenital heart defects indicates the need for widely applicable techniques for surgical correction of these anomalies The essence of wide applicability and effectiveness in almost any form of medical treatment is simplicity; and for that reason we have placed a sustained emphasis upon this requirement in the development of methods for intracardiac surgery, often discarding techniques that introduced complexities

In this regard, it might be mentioned that history has a strange way of repeating itself. It was Meltzer and Auer's[14] ingenuity in developing a safe and simple method for maintenance of the patient by means of an endotracheal tube during an interval of open thoracotomy, and not Sauerbruch's unwieldy pressure chamber, that sparked the subsequent rapid advances in intrathoracic surgery that have occurred.

SUMMARY AND CONCLUSIONS

1. Technical considerations in the clinical application of two methods permitting total by-pass of the heart and lungs for direct vision intracardiac surgery are presented.

2. These methods are: (1) controlled cross circulation, and (2) continuous arterial perfusion from a reservoir of arterialized-venous blood.

3. With the cross circulation technique there is a continuous reciprocal interchange of arterial and venous blood controlled by a pump between the patient and donor during the interval of total by-pass. The essential functions of the donor are to oxygenate and revitalize the venous blood received from the patient.

4. In utilizing the continuous perfusion method the general arrangements are the same as for cross circulation with the exception that the donor's place in the extracorporeal circuit is occupied by arterial and venous reservoir bottles. The former are filled with previously collected heparinized arterial blood, warmed to body temperature, and the latter are provided to collect the venous blood withdrawn from the patient during the perfusion.

5. For this procedure a significant contribution to the simplicity and convenience of collection of arterial blood in the blood bank by the ordinary phlebotomy technique has been the use of cutaneous heat applied to the arms of the donors to arterialize their venous blood.

6. By use of one or the other of these two techniques, 40 patients with ventricular septal defects, atrioventricularis communis, isolated infundibular pulmonic stenosis and tetralogy of Fallot lesions have had their heart and lungs totally by-passed for an interval of 5 to 40 minutes during which time a cardiotomy has been carried out permitting surgical correction of the intracardiac defects under direct vision.

7. The results in these patients are briefly reviewed. One or more patients in each of these four categories of congenital heart disease have been objectively restudied postoperatively by cardiac catheterization and demonstrated to have normal circulations.

8. The septal defects have been repaired by direct suture.

9. A blood-free intracardiac operating field has been obtained by a tourniquet about the base of the aorta to control selectively the coronary blood flow and aortic regurgitation

10. There has been no donor mortality in the cross circulation operations.

11 All of these 40 patients have been perfused during their total by-pass interval at substantially lowered rates of blood flow ($\frac{1}{4}$ to $\frac{1}{8}$ of the normal basal or resting cardiac output) based upon the azygos flow experimental studies. There has not been a single instance of detectable cerebral, hepatic,

renal or cardiac dysfunction attributable to these reduced perfusion rates at normal body temperature. The vast simplifications made feasible by these lowered flows have contributed importantly to the success of these methods.

12. Conduction difficulties have not been a serious problem in these intracardiac procedures because of close attention to two factors important for the well-being of the cardiac conduction system, namely, normal body temperature and a well oxygenated myocardium. It is emphasized that once the myocardium is relieved of its pumping burden, as occurs when venous inflow stasis is effected, it remains well oxygenated on a mere fraction of its usual coronary artery flow

13 The complete absence of such possible complications as air embolism, excessive hemolysis or postoperative hemorrhage has been gratifying

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Question: Would you be willing to apply the technique to mitral regurgitation? Also, what is the present upper limit of patient age for effective cross circulation?

Answer: We have thought somewhat about the problem, obviously. I have felt myself that the final solution of mitral regurgitation will depend upon a direct vision procedure, and I think one could possibly conceive of much easier ways of by-passing just half of the heart, using some of the principles enumerated here, than to totally by-pass it.

Obviously, to by-pass half of the heart would make it relatively easy to use the patient's own lungs, which I think is one of the stages that we have actually passed through in developing the cross circulation technique.

As some of you recall, we did not try it clinically because in dogs it was found that the lungs interfered considerably with the exposure for some of these complicated intracardiac defects, and if the pulmonary venous cannula became kinked for just an instant during one's attention to the intercardiac pathology, the lungs were full of fluid and the operation was over. Therefore, the cross circulation technique seemed much better because it obviated those difficulties.

I think it is an important lesson that was given to us, and I pass it on to you who are interested in the field, that the method for working within the heart has been far less of a problem to us than what to do after one gets into the heart, namely, recognizing the intimate details of the anatomy of these defects and knowing how best to deal with them

The other question concerned the upper age limit. The oldest patient we have operated on is a 14-year-old girl who weighed about 100 pounds

Some of you were at our institution last week and saw some of the enthusiastic surgical residents working in the laboratory. They have regularly been perfusing dogs on flows of 5 and 10 cc. per kilogram of body weight per minute. Applied to an adult, that is a relatively small quantity of blood. I would not hesitate to apply it to an adult.

At the present time we have not operated on any adults, which is merely a manifestation of the fact that most of these serious defects do not reach adult age. If they do, they reach the stage where their pulmonic flow in the ventricular septal defect group, anyway, is substantially less than the systemic flow, and I think they represent a considerably more difficult problem at that stage, because simple closure of the defect will probably not be compatible with life unless one can place an alternative shunt somewhere.



DISCUSSION

r **Angelo Riberi** (*Indianapolis*)

Working with Dr Shumacker, we have been studying the problem of the pair of ventricular septal defects Though our experience has not yet passed e experimental stage, you may be interested in hearing briefly about our ork with the closure of ventricular septal defects under hypothermia.

In a series of dogs protected by sino-atrial procaine blockade, we have been ble to produce and repair ventricular septal defects with survival of all of e animals. In 30 per cent, we encountered air embolism to the coronary rteries With a technique we have developed it has been possible to flush way the air from the coronary vessels, to increase the tone of the myo-ardium, to defibrillate the heart successfully and to resuscitate all of the nimals.

Whether the method will prove safe for humans we do not know Dr humacker did carry one child safely through the operation with closure of e defect, but death occurred some time later, possibly from cerebral air mbolism.

13. Lillehei, C W, Baronofsky, I D, and Varco, R. L · Surgical treatment of congenital heart disease Bull Univ Minn Hosp., *24*:75, 1952.
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CARDIAC ARREST

JOHN H GIBBON, JR (*Philadelphia*)—CHAIRMAN

CARDIAC ARREST

FRANK GERBODE (*San Francisco*)

There is no surgical emergency which requires more prompt action than cardiac arrest. It is an emergency which seldom occurs without, at least in retrospect, an ascertainable cause. "An ounce of prevention is worth a pound of cure" finds no better application than here.

The increasing scope of surgery, extending as it now does to include the tiny infant and the aged, has posed new hazards and difficulties for the anesthetist and surgeon and made the opportunities for the development of cardiac arrest greater than ever. Furthermore, the magnitude of excisional procedures has increased to the point that one often wonders whether we are not at times exceeding the natural boundary of extirpation. The frontier in cardiac surgery continues to pose problems in physiology which occasionally exceed our capacity to cope with them, leading to instances of cardiac arrest and ventricular fibrillation. However, one can safely state that as experience is gained with each procedure the number of unexpected emergencies decreases.

There are obvious causes of cardiac arrest such as severe shock and massive blood loss, the deleterious effect of torsion, displacement and manipulation of the heart is well known, and is increased when added to an already diseased myocardium.

Although a vast literature has accumulated on the causes of cardiac arrest, one can safely state that difficulties arising from inadequate ventilation of the lungs account for a great many, if not most, instances. Poor oxygenation resulting from an impaired airway or ventilatory exchange, insufficient oxygen administration or compression of the lung may set the stage, and some otherwise innocuous stimulus may cause cessation of cardiac function

Carbon dioxide retention, incident to inadequate ventilation, causes respiratory acidosis, and perhaps by the release of phosphoric acid into the blood a metabolic acidosis as well If acidosis is prolonged potassium concentration may rise, and exert a deleterious effect Hypercapnea is more serious in the presence of cardiac disease. There is experimental and some clinical evidence that in the presence of severe respiratory acidosis ventricular fibrillation may result from sudden ventilation with a mixture low in CO_2. Since inadequate alveolar ventilation is the most common cause of CO_2 retention and acidosis, it is obvious where attention must be paid during general anesthesia. I believe these are factors which favor the use of endotracheal anesthesia

There are many causes of operating room deaths which should not properly be included in the usual category of cardiac arrest. Among these are uncontrollable shock, massive blood loss and cardiovascular accidents.

The frequency of cardiac arrest depends on the condition for which the operation is being done. In general, it varies between one in 3000 and one in 5000 operations In certain types of surgery, such as cardiac, it is understandably higher At Stanford University Hospitals, during the past seven years, the incidence among noncardiac cases was one in 3673, and in the cardiac one in 41 As an example of the material from which these figures are obtained, during the academic year 1954 there were 4123 operations of all kinds performed under general anesthesia, and 2466 other operating room procedures, with 3 arrests. One service patient, who had a difficult esophagectomy, developed uncontrollable bleeding from afibrinogenemia, one elderly male developed uncontrollable bleeding during a transurethral resection, and one adult who was having a coarctation of the aorta repaired had an arrest which was successfully treated In this year there were 134 cardiac procedures performed.

There is proof of the deleterious effect of neurogenic influences upon the heart. Pain stimuli generally have an accelerating response or cause disturbances in rhythm. Vagal effect is more potent: it may arise from a hypersensitive carotid sinus, causing bradycardia, or by direct stimulation in the thorax, causing asystole. Anoxia and hypercapnea increase the effect of vagal stimulation. In this regard it is important to heed the appearance of bradycardia for it may precede cardiac arrest. The use of curare in combination with other anesthetic agents has been held responsible for an increase in anesthetic deaths, but there is no unanimity of opinion on this. Cyclopropane has been found to have a profound influence on cardiac function under certain circumstances, and when epinephrine is administered to a patient anesthetized with this agent there is danger of inducing ventricular fibrillation.

Under the direction of Dr Philip Bailey of the Department of Anesthesiology at Stanford University Hospitals particular attention has been given to increasing the safety factors in anesthesia, especially in poor risk and cardiac patients Many of these patients can be maintained on pure oxygen for operations lasting one to two hours if the following general plan is used.

1 Premedication with Demerol (maximum dose 100 mg) and scopolamine 0 4 mg
2. Induction (while being given oxygen) with Pentothal (maximum 0 5 gm.) (Pentothal is not used as the anesthetic.)
3 Spraying the trachea with not more than 3 cc. of 4 per cent cocaine (to lessen reaction to endotracheal tube)
4. Dimethonium bromide 3 mg
5 Controlled respiration with pure oxygen.
6 If patient moves, give small additional doses of Pentothal.
7 If necessary, use nitrous oxide for closure.

The use of hypothermia in cardiac surgery has made possible the closure of atrial septal defects under direct vision, is an aid in the resection of thoracic aortic aneurysms, and is helpful in certain other types of operations for cyanotic heart disease. It has also introduced the new hazard of ventricular fibrillation in the hypothermic state, which is as yet not completely understood or

hypothermia at body temperatures reduced to 28° C, but below this the dangers increase considerably Ventriculotomy under hypothermia almost invariably results in ventricular fibrillation, which usually is impossible to convert to sinus rhythm

RECOGNITION

Despite all that has been said about the recognition of cardiac arrest the most reliable sign is the absence of a pulse. Electrocardiography is very helpful but it is important to remember that it will not tell how efficient the heart is as a pump. It will differentiate between standstill, idioventricular rhythm and fibrillation; but very often the instrument is not being used when the unexpected emergency occurs, and then it is too late Prolongation of the QRS and T wave changes may herald more serious events Auscultation of the heart is also unreliable, for here again it is impossible to determine the true state of affairs If there is no pulse or audible heart sound one should not delay, but proceed at once with thoracotomy.

Observation of the heart during open thoracic procedures provides the surgeon with the best information obtainable about its status. Ventricular dilatation, abnormal rhythm or slow feeble contractions are ominous signs, and when they appear the operation should be temporarily suspended until the cause is found and corrected. The color of the blood is a fair indication of the cardiorespiratory status; if it does not appear to be satisfactorily oxygenated, surgeon and anesthetist should seek means of improving the situation It is not uncommon for the surgeon to recognize cardiac arrest before the anesthetist, for he is often in a better position to evaluate the status of the patient.

TREATMENT

The most essential feature of cardiac resuscitation is prompt action, for a delay of 3 to 5 minutes may cause permanent central nervous system changes Treatment consists of (1) maintaining respiration with oxygen administration, (2) maintaining circulation with rhythmic compression of the heart and (3) applying the proper resuscitative measures to the heart

Control of respiration is an essential aspect of cardiac resuscitation, and if not already being maintained it should be established as soon as possible Pure oxygen is used, preferably through an endotracheal tube, but it may be necessary to use a tight-fitting mask Respiration is sustained through manual compression of the anesthesia bag. Such artificial respiration serves the double purpose of oxygenating the blood and aiding pulmonary circulation.

Although there are recorded instances of successful resuscitation of the heart through the intact thorax using thumping, and injections of epinephrine, it is not recommended. The question is also often raised as to the effectiveness of the transdiaphragmatic approach through the abdomen. This has the disadvantage of not exposing the heart for direct observation and proper treatment, furthermore, an open thorax is usually obtained anyway, and this must be treated

The preferable approach is through the fourth interspace anterolaterally,

with incision of the fourth and fifth costal cartilages One should not waste valuable time searching for antiseptic solutions or special instruments Possible infection is a secondary consideration, and a knife alone will open the thorax.

The pericardium should be opened, for one can compress the heart better directly than over an intact pericardium. This also permits inspection, answering the question of whether standstill or fibrillation is present. Furthermore, the proper site for drug injection may be chosen. Our preference is the right ventricle or pulmonary artery, for the drugs will reach the coronaries more steadily after passing through the lungs Others prefer the left ventricle, believing that prompt filling of the coronaries with the drug is important. Either epinephrine or calcium chloride solution is injected if standstill or feeble pulsations are present. In most circumstances it is best to administer a rapid infusion of blood, or until available, glucose or plasma expander The use of the Trendelenburg position is advantageous Rhythmic compression of the heart is begun at once, across both ventricles, with minimum cardiac displacement. Compression of the heart from below against the sternum is a satisfactory method for small children and infants, but in an adult it usually causes too much displacement and is ineffective. A compression rate of 60 to 80 per minute is maintained. The anesthetist may be helpful by observing whether the method being used is causing an effective carotid pulse. Clamping the aorta beyond the left subclavian will force more blood to the brain and coronaries, but it must also be remembered that this deprives the spinal cord of blood, which after a time will cause permanent changes there.

Ventricular fibrillation is treated most satisfactorily by defibrillation with an electrical defibrillator All hospitals where anesthetics are given or operations performed should be equipped with one. It must be remembered that rhythmic compression should be applied to the fibrillating heart while preparations for electrical defibrillation are being made, since coronary and cerebral circulation needs to be maintained. A single shock across both ventricles of 110 to 130 volts (1.5 amperes) for 0 1 second duration is first tried, and if unsuccessful in producing arrest or return to normal rhythm then repeated shocks should be tried. Good electrical contact with the myocardium through gauze moistened with saline solution insures an adequate passage of current through the heart. It is important to remember that weak shocks (0.5 ampere) cause fibrillation. In general, the sooner electrical shock is applied the better is the prognosis

Injection of procaine (10 cc. of 1 per cent solution) or procaine amide (100 to 200 mg) should be used only if defibrillation is difficult of accomplishment. These drugs depress the heart and circulation. If the heart is flabby and soft, epinephrine (0.5 cc. of 1 1000 solution in 10 cc. saline) should be injected into it for it is often impossible to defibrillate an atonic heart. After injection, continue rhythmic compression until it becomes firm and the fibrillation becomes seething and turbulent, then defibrillate.

If there is a correctable surgical lesion present such as a valvular pulmonic or mitral stenosis, this should be treated quickly during the early resuscitative period, for massage is ineffective until this is done.

The question frequently arises as to when resuscitative measures should be abandoned. Some individuality must be used here, but it is a recorded fact that the heart has been revived after rhythmic compression of the heart has been maintained for nearly two hours. One is more apt to continue resuscitative efforts if the heart is a comparatively healthy one, than if obviously badly diseased.

Electronic pacemakers which have been devised to take over and maintain the heart beat are still in the experimental stage and not ready for general clinical use for the treatment of cardiac arrest.

USEFUL DRUGS

A vast literature has accumulated on the uses and actions of the many drugs which have been suggested for cardiac resuscitation. Space does not permit of a full discussion of all these agents, nor would it be of value, for only a few have been found to be essential. Epinephrine 1 : 1000, 0.5 cc. in 10 cc. isotonic sodium chloride solution, or 2 to 5 cc. of 10 per cent calcium chloride solution is the drug of choice in treating standstill or feeble pulsations, and generally speaking calcium should be tried first Procaine (10 cc of a 1 per cent solution) or procaine amide (100 to 200 mg) should be used if electrical defibrillation is difficult to accomplish, or if the heart is excessively irritable, but as mentioned above these drugs depress the circulation and seldom are used.

Epinephrine is a very potent cardiac stimulant which directly affects the myocardium, causing an acceleration in the rate. In the heart sensitized with cyclopropane it may precipitate ventricular fibrillation. Norepinephrine exerts an excitatory action which may also lead to ventricular fibrillation. Ephedrine acts much like epinephrine, but with diminished intensity. Generally speaking, the same can be said of Neo-synephrine and amphetamine. Calcium chloride has long been known to increase ventricular contractibility, it is used to overcome standstill, to counteract the effects of procaine, and in general when an increase in irritability of the myocardium seems desirable. Barium has a similar action, but is less constant in the response it causes The other stimulants, such as caffeine, cocaine and picrotoxin, have no place in resuscitation of the heart under these circumstances. Atropine is considered less efficacious than procaine or procaine amide as a blocking agent.

Potassium chloride intravenously in 0 5 per cent solution has been advocated to counteract ventricular fibrillation caused by electric shock. It accomplishes this by depressing the conductivity of the heart, but on the whole it is not a very satisfactory drug to use Excess or an overdose of potassium is neutralized by calcium. Quinidine has no use in the acute situations found in the operating room Under experimental conditions it has been demonstrated to make ventricular fibrillation harder to produce, but we have not found it useful, even as a prophylactic drug.

PROGNOSIS

The prognosis depends upon the duration of the arrest, the duration of the resuscitative procedure and the condition of the patient. Negroes and other

dark-skinned individuals have a poorer prognosis than whites Many patients have had successful resuscitative procedures after 2 to 5 minutes, only to die hours or days later A great limiting factor is the vulnerability of the central nervous system to anoxia, which places a limit of 3 to 5 minutes on the duration of cessation before damage occurs There is some variation among patients in this regard. It has been our impression that patients who have had a small cardiac output for a long time, such as those with mitral stenosis, will tolerate cardiac arrest for a longer period of time than those who have previously had a normal cardiac output.

Neurologic changes resulting from cardiac arrest vary from minimal signs to decerebration. Blindness, deafness, ataxia, speech disturbances, apathy and mental deficiency have all been reported, the results of degenerated central nervous system. Nevertheless, it is important to remember that remarkable clearing has occasionally taken place. Of 1200 cases of cardiac arrest a permanent survival rate of 28 per cent has been recorded. It is apparently a fact that ventricular fibrillation has approximately this same prospect.

CONCLUSIONS

Cardiac arrest is the most urgent emergency one must face in surgery It is the responsibility of the physician in charge of the patient to institute resuscitative procedures at once, for the delay which follows when someone else is sent for results in the loss of the 3 to 5 minutes during which the central nervous system remains viable.

The subjects of cardiac arrest and ventricular fibrillation should be discussed openly in staff conferences, and the responsibilities and methods to be used should be clearly established.

When proper measures are instituted approximately one fourth of such patients can be revived.

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Question· What about aortic compression?
Answer. I can say that it will increase coronary flow, it will increase the effectiveness of the massage, but don't forget that if you have a clamp on the aorta you are shutting off the blood supply to the spinal cord, and so it is only a temporary expedient which would apply only for a short period of time I myself have not used it except in the laboratory.

Question· How does Dr. Gerbode arrive at his conclusion that multiple anesthetic agents are a cause of arrest?
Answer That is a question that very often arises, and I am in constant difficulty whenever I mention this, particularly in front of anesthesiologists I think this probably was asked by an anesthesiologist.

I believe we are all convinced that many of our patients have been plagued with too wide a variety of drugs during operative procedures. We stimulate them, we sedate them and we vacillate back and forth, and by the time we are finished very often the autonomic nervous system

is so confused that it doesn't know what to do I think Harry Beecher's very excellent survey of this subject will allow you to conclude that multiple anesthetic agents and also perhaps curare have had some influence on this However, I don't think we can lay the whole thing on the doorstep of curare, because some people have had excellent results with it.

Question Must the greater surface of the heart be covered by electrodes, and why?
Answer No, it need not be covered by electrodes We use an electrode about 2 inches in size. I think good contact is necessary, however, and the electrode should be covered with a sponge which is preferably soaked in saline, otherwise some burning of the heart will take place.

Question How do we define cardiac arrest?
Answer I think the best vital sign you can rely on is an absence of a pulse or absence of a heart beat. If you find that in your patient, then you must assume that the heart has stopped. You may find that it has not stopped if you open the chest, but it is better for you to get into the chest while the heart is beating a little feebly than to get in when it is not beating at all, or is fibrillating.

I think that is the moral of the story I don t believe it does any good to have an electrocardiogram brought in to find out whether the patient is dead or not. What you have to do is to decide that if there is no pulse and no heart beat that you can determine, there is a cardiac arrest and you must do something about it.

Question What do you think about intra-aortic transfusion of oxygenated blood during cardiac arrest?
Answer I think it is probably a very good thing to do if you are set up to do it, but again you would have to be prepared in advance. Most people aren't.



DISCUSSION

Dr Claude Beck (*Cleveland*)

The subject of resuscitation is in a rather different category from some of the other procedures that have been presented, because I think our knowledge of resuscitation is now more or less fixed and established I don't think we are going to be changing it from year to year with additional experience, and I believe the problem today is for surgeons to assimilate this knowledge and apply it properly at the proper time.

I have nothing but emphasis to add to what Dr Gerbode has already told us It is very important for us to divide the resuscitation procedure into two steps The first step is to reestablish the oxygen system. You have to

get oxygen into the lungs, and you have to circulate it to the brain, and you have only three, four or five minutes to do it Nothing else should enter your mind When the oxygen system has broken down you must reestablish it. That is a big enough job to do in itself, and you must not use your stethoscope too much; you must not call for a cardiogram; you must not call anyone, you have to take care of the situation yourself, and you have to do it right away.

How are you going to get oxygen into the lungs, and how are you going to circulate it? It is obvious that you have to have a tube ready to put into the trachea, and someone who is able to put the properly fitting tube into the trachea. You have to have a rubber bag and oxygen, and you have to squeeze the bag. The equipment ought to be available at the head of every patient in the operating room The chief of surgery ought to make it a standing rule that this emergency equipment is always at the head of the patient, whether general anesthesia or local anesthesia is being used.

After the oxygen is gotten into the lungs you have to circulate the oxygen, because if it is just in the lungs it does not do any good. Is there any way to circulate it? The way is to slit open the chest and get your hand in there and squeeze the heart. You are not going to take time to open the pericardium. You are going to grasp the heart and empty it, and it is advisable for every surgeon to have some experience in squeezing the heart so that he won't squeeze an empty heart and he won't traumatize the heart unnecessarily.

After you have the oxygen system started, then the crisis is over and you can take a few breaths on your own, and quiet down a bit, but up to that time you have to move, and move rapidly, and you can't waste a moment. You can't even sterilize the skin—you can't take any unnecessary step.

The second step of the procedure is the restoration of the heart beat. Dr. Gerbode spoke about that I would like to say only that any normal heart can be made to beat again if properly handled. I think it is up to us surgeons to see to it that we do know how to do it properly.

The third thing I want to mention is the responsibility that we surgeons have to society for resuscitation. This knowledge of resuscitation has been in existence for a good many years, and, if I may say so, I think we have been a bit derelict in picking it up Just within our family, I would like to say that we really should not operate on a patient unless we know how to resuscitate his heart if it should stop.

I don't think we are doing the right thing for our patient unless we have knowledge of resuscitation procedures. I think it is a crime for a surgeon to walk away from the operating table, the patient dying, without doing anything. If I were on a jury in a court and had to make a decision about the responsibility for the death of a patient with a previously normal heart who died on the operating table, I would have to vote against the surgeon

There is no excuse, it seems to me, for a surgeon not knowing how to resuscitate a heart. Courses have been established in various parts of the country to teach surgeons and anesthetists how to do it. It is exceedingly simple We ought to avail ourselves of those courses. Also, we ought to have a "fire drill" in every hospital once or twice a year. The whole subject should

be gone over by the staff, preferably we ought to expose a dog's heart on a Sunday morning when nobody is around except the staff, and go through the exercise of resuscitation, fibrillation and defibrillation over and over again so that everyone will feel perfectly familiar with doing resuscitation.

We have been giving our course for about five years, and I suppose we have had about 750 surgeons and anesthetists take it. I can't give the figures accurately, but I have a feeling that there have been about 75 instances in which surgeons have written in saying that they have successfully resuscitated a patient with the knowledge they have gotten from the course.

The only thing I can add to Dr Gerbode's excellent presentation is to urge the surgeon to feel that he has a responsibility in doing this job correctly We ought to be far advanced from the stage when the surgeon called the fire department for a pulmotor to help him out.

PANEL DISCUSSION ON ADVANTAGES AND DISADVANTAGES OF VARIOUS METHODS OF INDUCING HYPOTHERMIA

WILFRED G. BIGELOW (*Toronto*), CHAIRMAN

SIR RUSSELL BROCK (*London*)
DENTON COOLEY (*Houston*)
CHARLES DUBOST (*Paris*)
STERLING EDWARDS (*Birmingham, Ala.*)
HELMUT JAEGER (*Santiago, Chile*)
JOHN LIND (*Stockholm*)
HARRIS B. SHUMACKER (*Indianapolis*)
HENRY SWAN (*Denver*)

DR. BIGELOW

At the present time there are several different techniques for cooling, both experimentally and clinically Dr Swan, would you start this out by enumerating some of the methods in current use? You might indicate which technique you use, and why you prefer it

DR SWAN

Basic to all techniques is the induction of anesthesia or suppression of the nervous system in order to have the individual free of muscular activity, so that the body may cool more readily without the production of a strong reaction on the part of the individual being cooled

The application of cold can be done in a large variety of ways If one thinks of surface cooling, one thinks usually of the external surface of the skin of the body, and to the skin of the body can be applied cold air, cold water or ice in various forms. Also, we may use mechanical devices with refrigerating units and rubber blankets in which a refrigerant circulates.

One can also use some of the internal surfaces of the body, such as the pleural and peritoneal spaces, which can be subjected to a cold agent

The more direct approach is to take the blood out of the body and cool it by some sort of mechanical refrigerant. One can take blood out of a vein and put it back into a vein, or one can take blood out of an artery and let it go back into a vein. The cooling, therefore, is extracorporeal.

Our particular method of cooling was adopted because it was simple, inexpensive and rapid We did not know whether it was desirable to cool rapidly or not, but we had enough distrust of this unknown physiologic state of general hypothermia to wish to get the patient into a cold state, achieve the purposes that we had in mind in placing him there, and then to warm him up again as rapidly as possible.

For this reason, we use a maximum amount of body surface, that is, almost the total skin area, we use the best known conductor of heat, namely, water, and we use ice water This technique is rapid.

DR. BIGELOW

Dr Lind, will you tell us what technique you use?

DR. LIND

I have worked only with dogs in experimental hypothermia, and we have used only ice water, shaving the dogs beforehand. We have found that the cooling time depends on the size of the dog In doing a large series of dogs with Dr Juvenelle, from Paris, we found that cooling time by the usual hypothermic experiments had no significant relationship to the later survival of the animals

SIR RUSSELL BROCK

I asked if I could have the privilege of saying a few words about a different method of cooling—blood stream cooling—that I have used.

I believe I am correct in saying that in the United States almost all the work has been done with surface cooling. We have used two methods in my clinic. First of all, arteriovenous cooling was introduced, and we found that there were certain disadvantages in it, notably that the heart itself, being the pumping agent, began to give trouble as the blood pressure and circulatory efficiency fell with the rate of cooling That was particularly so in cases with a heart lesion and with a shunt of some sort.

Then we went to venous cooling That has certain advantages. To begin with, we took blood for cooling by putting a catheter in through the external jugular vein, passing it down into the superior vena cava, conducting the blood through a cooling coil, a plastic tubing immersed in a refrigerant, and then putting it back onto the femoral vein.

We had some trouble with children in getting into the superior vena cava properly owing to small neck veins, and the next step was to open the chest and put the catheter into the heart. That introduced what I think is a very helpful principle, namely, we cannulate the saphenous vein first. Then we open the chest in the ordinary way at normal temperature. That enables one to investigate the condition and confirm the diagnosis Then one can put a catheter into the superior vena cava by way of the right auricular appendage, and start the cooling

The cooling takes from 20 to 30 minutes, and during that time you can do the various things—preparation of the vessels, and so on—which are necessary I might say also that at the end of the procedure one can reverse the method and start warming

Figure 1 illustrates the set up, the catheter via the right auricular appendage into the superior vena cava—a plastic tube through a simple hand pump and then through a cooling coil. The refrigerant is circulated in the surrounding reservoir from a modified household refrigerator The cooled blood returns to the inferior vena cava.

The catheter is withdrawn from the superior vena cava into the atrium immediately before the great veins are occluded, with the advantage that if you continue pumping you can suck the heart dry, or partly dry, and therefore make the opening in the heart easier.

In the principal arteriovenous cooling as introduced by Delorme and Boerema, the femoral vessels are exposed in the femoral triangle and cannulated. The cooling sheath extends down almost to the artery itself. Cold

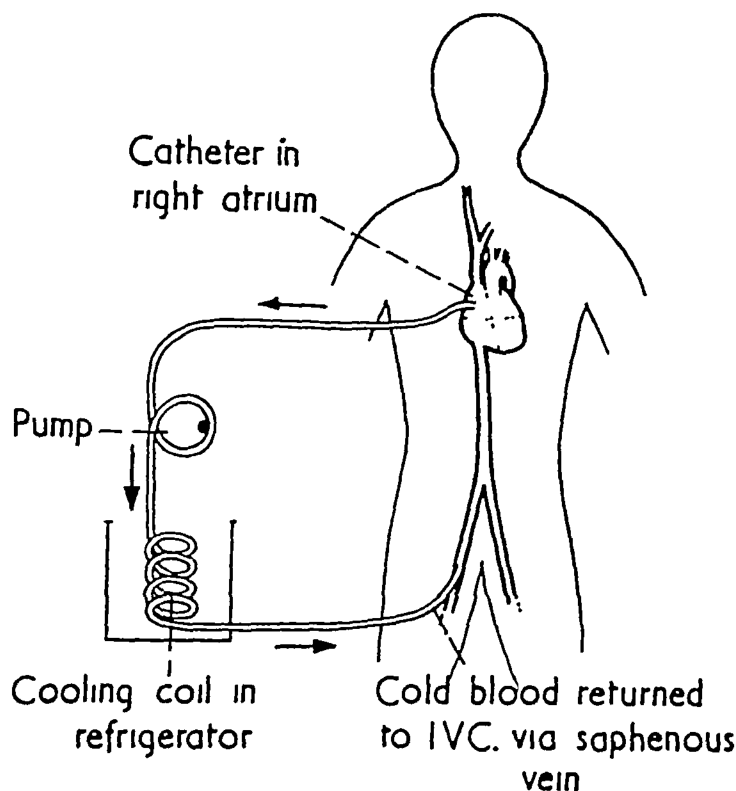


Fig 1. Method of veno-venous cooling. Catheter is placed in femoral vein before thoracotomy and second catheter in superior vena cava via right atrium after the heart is exposed.

water is pumped into the sheath around the coil through which the blood circulates. This coil, of course, is immersed in a suitable refrigerant.

We found that, in addition to the disadvantages I have mentioned of the failing heart action, in children, you may not be able to repair the artery properly and may have to ligate it, or you may have a secondary thrombosis, and that is not good

A simple hand pump is used (Fig 2); an electrically driven pump would have some advantages

The cannula used for insertion into the heart is kept open by an ordinary saline drip. This is the tube going down to the saphenous vein. I should mention that one advantage of this technique is that you can put the saphenous vein cannula in place and use it for ordinary intravenous infusion If you are not sure whether you are going to use hypothermia or not, then you don't have to start cooling until you get the chest open. I had that problem recently when I was not sure whether I was dealing with an ordinary ductus or an aortopulmonary septal defect.

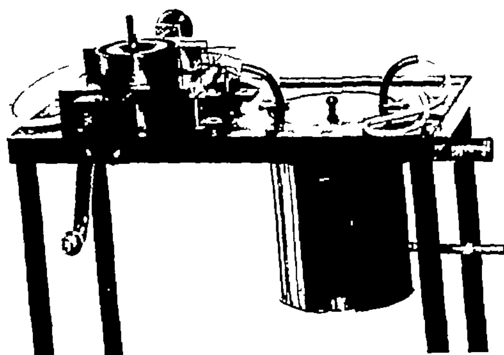


Fig. 2. Hand pump of roller type and heat-exchange unit through which the blood circulates in a coil and is either cooled or warmed.

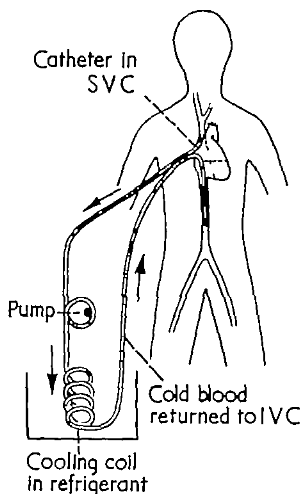


Fig. 3 Present method of veno-venous cooling, which can be decided on and used when the chest has already been opened.

After the operation you can reverse the process, and up to the time of closing the chest you can get 20 to 30 minutes of warming, which will warm the patient 3 to 5 degrees, which is a very great help.

Another great advantage is that you have the heart under constant observation, and you are able to resuscitate it immediately which is a little easier than jumping the patient out of the bath tub.

NOTE: Since delivering this account, we have simplified the procedure further by introducing *both* catheters via the right atrium, one into the superior and one into the inferior vena cava. Thus no preoperative cannulation of the saphenous vein is needed

DR. BIGELOW

Would you say that your chief advantage is the fact that you are prepared to use hypothermia if necessary, but you don't involve the patient in low body temperatures when it isn't necessary?

SIR RUSSELL BROCK

Oh, no, I don't think that is the chief advantage. That is one advantage.

There are certain objections to surface cooling, which you will admit or not admit. One objection that I have is that it is esthetically unattractive. I am full of admiration for the efficient results of the immersion techniques. They speak for themselves, but they do not seem right in an operating theater

DR. BIGELOW

Sir Russell, what about the cold blankets, which are somewhat more esthetic? I agree hypothermia should be as palatable as possible.

SIR RUSSELL BROCK

I am not trying to sell this, I am putting it forward for information

DR BIGELOW

Do any members of the panel wish to comment on that very interesting technique? We are interested in this because it is not experimental. We know Sir Russell has used it in clinical cases with success, and men who work with him like the technique very much

DR. SWAN

I have never tried to sell the bathroom scene in the operating room on the basis of its esthetic value, although actually sometimes these young patients are not offensive to be seen. On the other hand, I agree with Sir Russell that his is a more surgical procedure, that is, it involves a considerable amount of surgery to do it.

It is often necessary, in part of the total procedure of hypothermia, to indulge in a considerable amount of other forms of surgery, namely, that for which you took the patient to the operating room. Also, in our experience we have found it desirable to obtain direct reading of central venous pressures in order to be able to evaluate the patient through the course of the surgery.

Since we began working in this field we have felt that simplification was very important. We have tried to avoid the use of multiple medications We have tried to avoid the use of any activity which was not completely essential. For this reason, Sir Russell, I would say that my only objection to your very effective way of cooling might possibly be that you have added another set of tubes and incisions, and have injured some vessels perhaps unnecessarily

DR. COOLEY

I should like to cite one experience which should support the method which Sir Russell has proposed and which he has used so nicely

We had a patient who at least one hour after operation was still in a hypothermic state and was back in the recovery room when he developed ventricular fibrillation with circulatory arrest. An attempt to rewarm the patient with absent peripheral circulation was unsuccessful. Furthermore, it was impossible successfully to defibrillate the heart in the presence of hypothermia. Rapid rewarming of the patient is important because if complications are going to follow the operation itself, the surgeon can more effectively deal with them in a patient at the normal body temperature.

DR. SHUMACKER

I should like to add another way in which the method might prove useful. Although one might choose to use a somewhat simpler method of cooling, in general it would be very useful to have available some readily applicable rapid method of inducing hypothermia during the course of an operation. One will occasionally run across a patient in whom hypothermia will not have been induced and in whom it will seem desirable or necessary after the thoracotomy is performed. There it would be a great advantage to be able to induce hypothermia under such circumstances

DR. BIGELOW

This is specifically along the line of our discussion. How long does it take to cool to 30° by the various methods outlined?

SIR RUSSELL BROCK

Twenty to thirty minutes.

DR. SWAN

An 8 lb baby will cool to 30 in about ten minutes A 190 lb adult may take as long as an hour and ten minutes.

DR. BIGELOW

I don t know if any representatives on the panel do blanket cooling. I might say that Dr Mustard and Dr Keith, at the Sick Children's Hospital, use blankets and cold water In our adults we are as esthetic as possible, and we have not left the blanket technique. We have developed that to a point where we can cool adults to 30° in one and a half to two hours.

DR. STERLING EDWARDS

Just a word about rewarming. I agree with Dr. Cooley that it is probably wise to try to rewarm as much as possible before closing the chest. At least one will not get into the difficulties of which he spoke. We have tried to do this by leaving the chest open and pouring in warm saline, leaving it in the chest until the temperature has risen somewhat and the heart is beating more vigorously.

DR. SHUMACKER

I have been extremely disappointed with regard to the effectiveness of warm saline solution in the thorax as an agent for rewarming. When Dr. Blades and his associates reported this method it filled me with hopes that have not subsequently been realized.

DR. BIGELOW

Would someone like to answer this question? Do you believe rapid rewarming is preferable to slow rewarming, particularly in patients undergoing cardiac operations?

DR. COOLEY

I have already alluded to one experience which indicates that rapid rewarming is desirable. In my opinion the patient should be rewarmed as quickly as possible without actually producing surface burns. Recently, we have found that the Hubbard hydrotherapy tank in our physiotherapy department is a convenient and effective means of rewarming the patient. The water in the tank is maintained at 110 degrees F. and is continuously circulated over the body. The patient can be removed from the tank when his body temperature is 96 degrees F and then by means of wool blankets, his temperature may be allowed to rise gradually to normal levels.

DR. BIGELOW

At what body temperature are you so concerned that you are anxious to accomplish quick rewarming?

DR. COOLEY

Cardiac disturbances are more likely to occur at the lower levels in the hypothermic range, of course, and I prefer to get the body temperature above 91 degrees F. as quickly as possible without burning the patient.

DR. DUBOST

I have observed several burns on the back of a patient after rewarming in a blanket. I would like to ask Dr Swan what his experience has been with accidents when rewarming with warm water.

DR. BIGELOW

What has been your experience with warm water, Dr. Swan? There certainly is a danger in warm blankets

DR. SWAN

We have never had any difficulty with warm water In the last 40 to 50 patients we have used a diathermy coil around the pelvis of the patient, with a view to accomplishing what Sir Russell mentioned, namely, the ability to warm the patient with some facility in case we get into trouble with the heart beat and wish to warm the heart and the body in order to combat arrhythmia.

Therefore, we take the attitude that it is desirable to warm rapidly up to a point where the blood pressure is obtainable, the pulse is of good quality, and the heart beat is quite regular After that, slow warming is satisfactory

DR. BIGELOW

Roughly, what is that temperature?

DR. SWAN

In my opinion that temperature is 31° plus or minus 2 degrees.

DR. BIGELOW

Dr Jaeger, you have had experience with one of the techniques discussed Would you comment on the cooling or rewarming, or both?

DR. JAEGER*

Looking for an easy and inexpensive procedure to induce hypothermia, we first tried a modified Blades pleural cooling method Later it occurred to us to induce hypothermia by perfusing a cool solution through the peritoneum.

METHOD Mongrel dogs were anesthesized with intravenous hexobarbital soluble, and hyperventilated with oxygen through an endotracheal tube.

Pleural cooling After induction of a pneumothorax, two mushroom catheters (Malecot) were introduced with a Coryllos trocar in the left pleural cavity, one high up and anterior, to function as inlet tube, and the other low and more posterior, for use as outflow tube (Fig 1) Physiologic saline was used as irrigation fluid and cooled, running it through a copper coil, immersed in ice water The fluid collected from the pleural cavity in the lower bottle was recirculated, pumping it up in the upper reservoir

After the temperature reached the desired level, a fourth interspace bilateral thoracotomy was performed. The azygos vein was ligated, both venae cavae and the aorta were occluded for from 6 to 9 minutes. Ventricular fibrillation was prevented or treated with coronary perfusion of neostigmine, and electrical defibrillation. For rewarming, the same fluid was used, putting the copper coil in water at 45° C.

Every few minutes an ECG was done.

Peritoneal cooling A mushroom catheter (Malecot) was introduced with a Coryllos trocar high in the left flank, to function as the inlet tube, while a sump drain was introduced in the right lower quadrant, for use as the outflow (Fig 2)

* Aided by a grant from the W K. Kellogg Foundation.

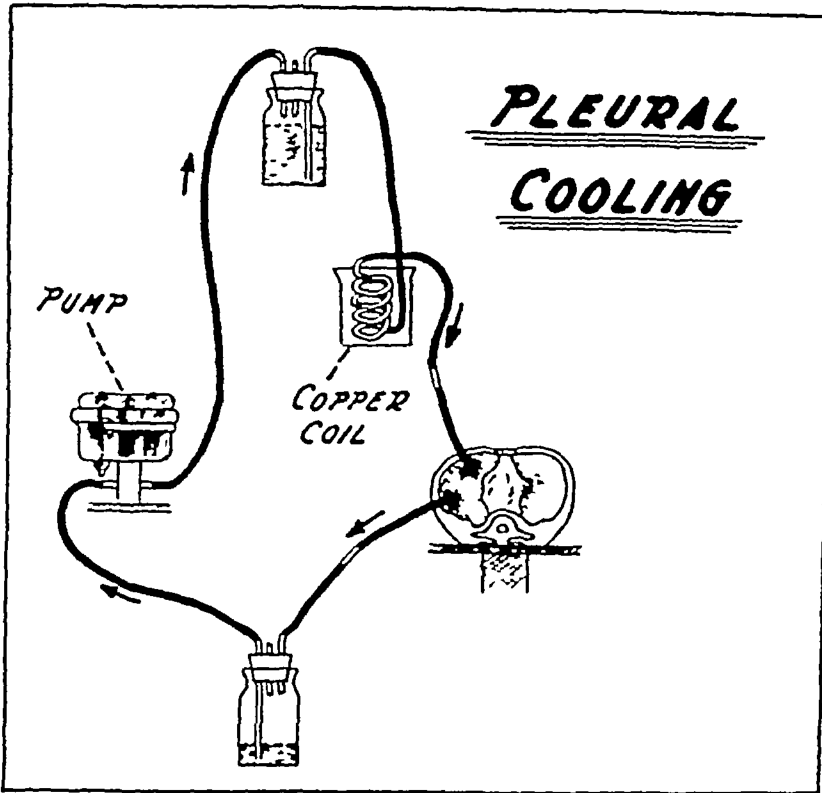


Fig 1 Device for pleural cooling

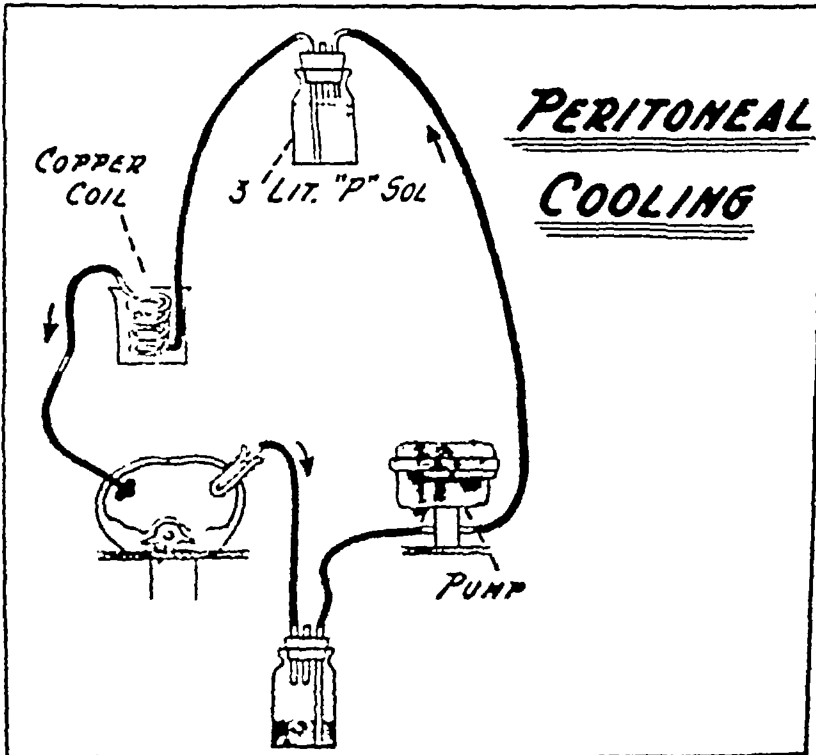


Fig 2. Set-up for peritoneal cooling.

"P" solution, as used by Odel et al. (1948) (Table 1) for peritoneal lavage in uremia, was used as irrigation fluid, and cooled as in the pleural method Only 3 liters of "P" solution were used, and continuously recirculated through the closed system.

A fourth interspace bilateral thoracotomy was performed. After the pericardium was opened, simultaneous rectal and intrapericardial temperatures were taken every few minutes Circulation was occluded as described before.

For rewarming, the same 3 liters of "P" solution were used, putting the copper coil in water at 45° C.

RESULTS. *Pleural cooling* Results have been almost identical to those originally described by Blades Temperature dropped about 1° C. every 5 minutes and was lowered to 24 to 27° C.

TABLE I "P" SOLUTION (Odel, et al., *M. Clin. North America*, *32*:989 1948)

Sodium chloride ($NaCl$)	6 0 g./liter
Potassium chloride (KCl)	0.2
Calcium chloride ($CaCl_2$)	0 1
Magnesium chloride ($MgCl_2$)	0 1
Sodium acid phosphate (NaH_2PO_4)	0 05
Sodium bicarbonate ($NaHCO_3$)	3 0
Dextrose	20
Penicillin	10 000 U
Streptomycin	20 mg.
Heparin	1 mg.

The *p*H of the solution is about 8 4 adjust to 7.5 by addition of chemically pure citric acid.

ECG alterations appeared at an average temperature of 31.5° C. (35 5–29° C.).

Peritoneal cooling The method was employed in 12 mongrel dogs weighing from 7.5 to 30 kg

The rectal temperature dropped an average of 1° C. every 5 minutes After cooling was stopped, there was no further drift of the rectal temperature. To reach this temperature it was necessary to run 2.5 to 8 liters of *cooled fluid through the peritoneum, recirculating the 3 liters* The use of the same 3 or less liters of solution should prevent an electrolyte or osmotic imbalance. A fatal pulmonary edema appeared in one dog, in which physiologic saline was used as cooling fluid.

When the solution was running fast through the peritoneum, the intrapericardial temperature was 1 1 to 4 4° C. lower than the rectal temperature. A few minutes after the cooling was stopped, the intrapericardial temperature began to rise, and reached the level of the rectal temperature.

The ECG showed about the same alterations as during pleural cooling.

During rewarming, rectal temperature rose an average of 1° C. every 8 minutes. The intrapericardial temperature was often 0 6 to 1.2° C. higher than the rectal temperature.

One dog died 48 hours after the procedure, from an intestinal hemorrhage. Autopsy showed the colon filled with blood and small mucosal erosions on the ileocecal valve.

In one dog, sacrificed 48 hours after the procedure, some parts of the small intestine showed a punctated subserosal hemorrhage and an edema of the mucosa.

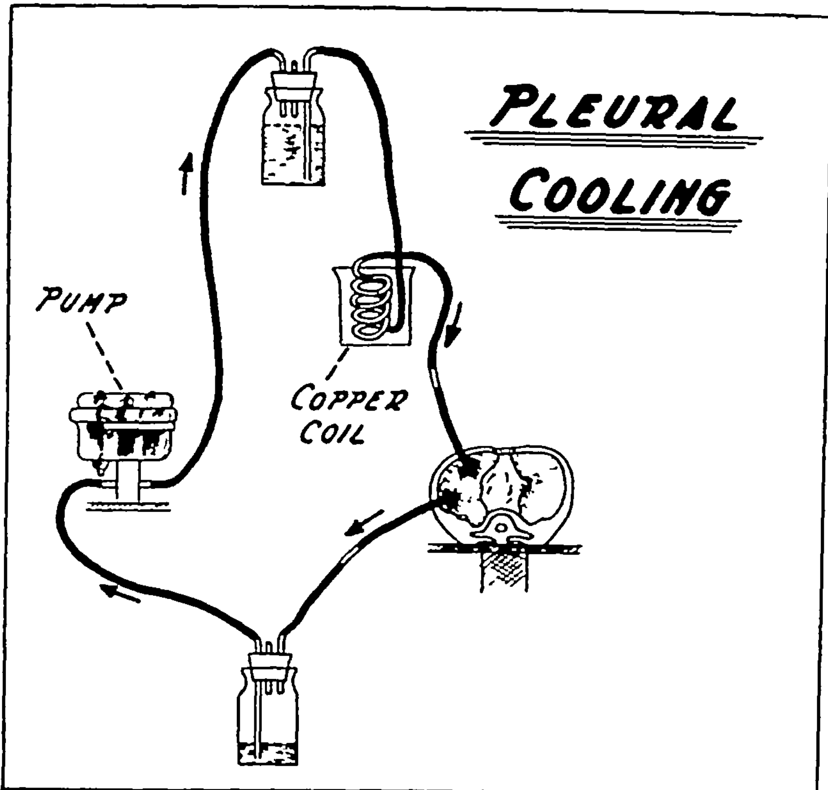


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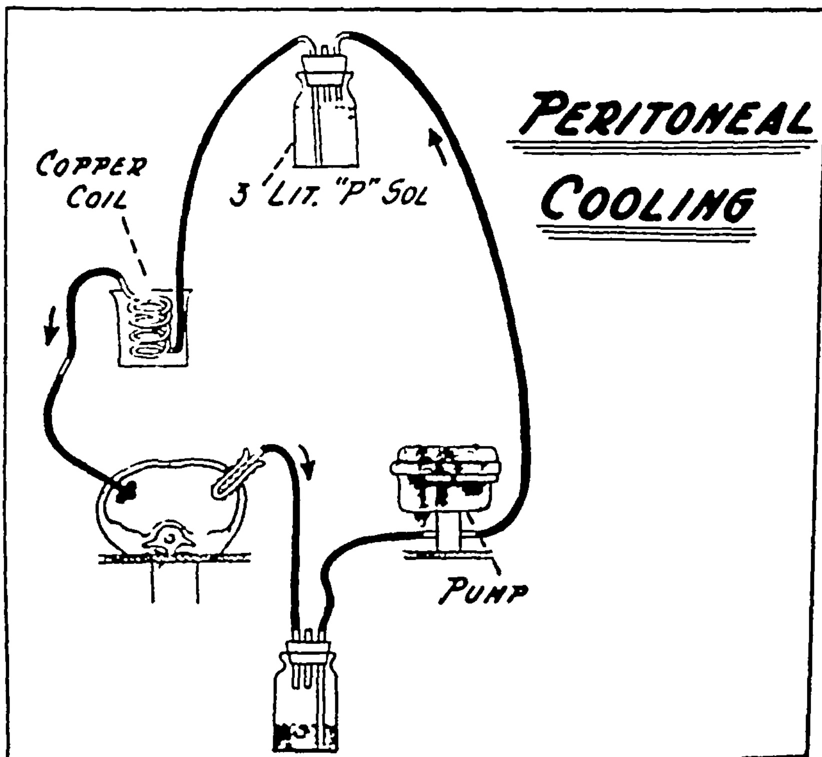


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In one dog, sacrificed 48 hours after the procedure, some parts of the small intestine showed a punctated subserosal hemorrhage and an edema of the mucosa.

COMMENT. The cooling time, with pleural or peritoneal cooling, is at least as fast as with the other methods.

With the use of mushroom catheters for induction of cooling in the pleural method, it is not necessary to have the chest open the whole time, as with the method first described by Blades.

The *peritoneal method* has the advantage that during cooling or rewarming the fluid does not hinder the opening or closing of the chest wall or the performance of intrathoracic surgery. Work is going on to find out if it is possible to prevent intestinal damage.

DR. BIGELOW

It is obvious, as is only correct in a new field such as this, that many methods are under investigation, and it will only be with the passage of time that we will find which method we should adopt.

Also, the methods of rewarming are variable, and some using hypothermia clinically seem to be afraid of maintaining patients at the temperature at which they are working for too long. I think there is evidence that one should not keep the temperature that low any longer than absolutely necessary. Only time will tell.

Sir Russell, is there any drift following cessation of direct blood cooling, and do you believe you have greater control of the hypothermia with your methods?

SIR RUSSELL BROCK

There is no drift in the experimental animal with intact septums. In the human with septal defects there is some degree of differential cooling, which on the whole is not much more than 1 or 2 degrees. The control certainly is greater, in my opinion.

DR BIGELOW

Dr. Cooley, in the case of ventricular fibrillation described, was the endotracheal tube taken out of the patient before the arrest occurred?

DR. COOLEY

No, the endotracheal tube was in place As a matter of fact, the patient was still in the recovery room and respiration was being continuously supported by the anesthetist when the cardiac arrest occurred.

DR. BIGELOW

On what physiologic basis was the body temperature of 30° decided upon? Dr. Swan, 30° is a fairly common temperature. I think the opinion of many who are using it extensively is that a fairly safe range of temperature is 28° C to 31° C, and at 31° C the oxygen demand is roughly 55 per cent of normal. When one wishes to interrupt the circulation, I think temperatures below that level are acceptable. You accept the added risk.

Dr. Swan, would you carry on from there? What is your opinion of the

temperature level, and on what basis do you decide to use any particular level?

DR. SWAN

The purpose for which we have used hypothermia has been based upon lowering the oxygen needs of body tissues I think the temperature chosen must depend upon the duration of time that you propose to withhold circulation from a part or from the body as a whole.

Our experimental evidence in the dog was based largely on a 25° dog with total cessation of circulation for 15 minutes. This is a very standard preparation in our laboratory, and we have very good results now as far as survival is concerned, without tissue damage in those animals

You get a slow and progressive addition of hypothermic effects as you go down at least to 25° We do not have very much experience below that figure. Therefore, if I wished to occlude the circulation for 3 minutes, I think 30° would be a very safe temperature to use. If I wished to be prepared to occlude it for 12 minutes, I believe I would go lower, and I would wish to have the patient at 25°

The difference in terms of safety in our experience is that cardiac irregularities are not common above 28° In the human one frequently sees atrial fibrillation above this temperature, but a serious arrhythmia is uncommon above 28° As you progress below that, the risk of this complication becomes progressively greater

DR. SHUMACKER

Someone should mention the fact that when one cools by external cooling to 30°, the temperature drops an additional several degrees as one prepares the patient. None of us has mentioned that so far I would say the drift would be 3 or 4 degrees.

DR. SWAN

We are talking here about lowest temperature change.

DR. BIGELOW

Even if you discontinue cooling and allow it to drift, it is the temperature at which you hope to operate.

Dr Cooley, do you have anything to say on temperature range in your aortic work?

DR. COOLEY

Our principal interest in general body hypothermia has been in its application to the surgical treatment of aneurysms involving the distal portion of the aortic arch and proximal descending thoracic aorta. Such lesions are best treated by segmental excision of the involved aorta and restoration of continuity by means of a homograft or cloth prosthesis The actual performance of this technical maneuver requires that the aorta be temporarily occluded above and below the lesion, usually for periods between 30 and 60 minutes,

and occasionally longer. The damage produced by ischemia to tissues supplied by vessels originating distal to the clamp may be serious, especially in those which are particularly vulnerable. Experience with 19 cases in which temporary occlusion of the thoracic aorta was necessary for periods of 24 to 60 minutes indicates that the spinal cord is the structure which is most susceptible to damage under these circumstances, and that the function of vital abdominal organs, such as the kidney and liver, is not significantly affected. The critical period of tolerable temporary interruption of cord cir-

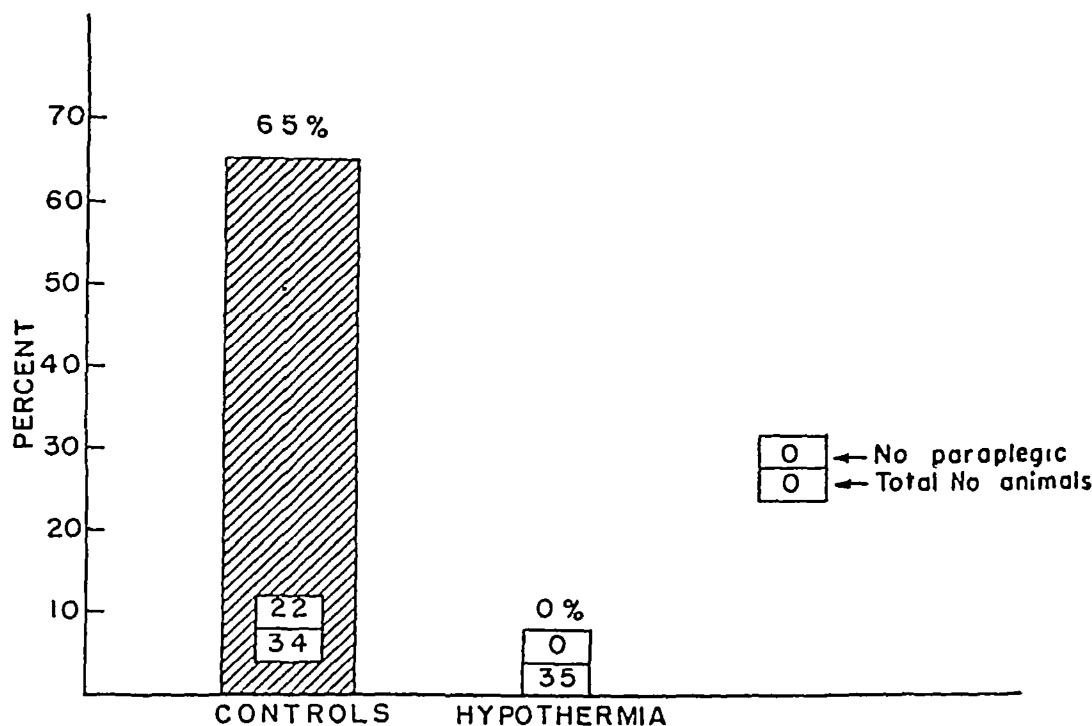


FIG. 1. Incidence of paraplegia in animals surviving aortic occlusion (From Pontius and others Surgery, vol. 36, 1954)

culation is not known but may be about 20–30 minutes. Long periods of aortic occlusion near the origin of the left subclavian artery may lead to paraplegia and even death. In order that this critical period may be prolonged, hypothermia has been employed to reduce the metabolic requirements of the cord.

In a series of animal experiments employing hypothermia, Dr. Robert Pontius who was working in our laboratory demonstrated that hypothermia is an effective means of controlling paraplegia after aortic occlusion (Fig. 1).

We have used hypothermia clinically in 7 cases of aneurysms of the upper thoracic aorta with periods of occlusion up to one hour, and none has developed evidence of neurologic damage. In 4 other cases with lesions located at the same aortic level, and in which hypothermia was not used, there were 3 in which definite evidence of cord damage appeared after operation. These changes were mild and transient in 2 cases but may have contributed to the death of the third patient

In our opinion, a hypothermic level of body temperature of 88–92° F. is

sufficiently low to provide the desired protective influence upon the spinal cord for aortic surgery In this relatively mild hypothermic range, most of the deleterious side effects of hypothermia may be avoided.

DR. BIGELOW

There is a question that naturally follows what we have been discussing It is actually directed to Dr Swan, so if he will lead off I think we will get a very rapid statement from the members of the panel. Is there any mortality inherent in cooling itself?

I think we might restate that for the benefit of the panel. Has anyone had cardiac arrest develop during cooling, before surgical procedure was started?

DR. SWAN

I will answer that question with an emphatic "yes" There is intrinsic danger in hypothermia, and it relates to the irritability of cardiac muscle in the cold state. We have had cardiac arrest. We have had ventricular fibrillation during the cooling procedure.

DR. SHUMACKER

We have not encountered difficulties before the operation was started, but I am sure the danger exists

SIR RUSSELL BROCK

Yes, of course we have seen it.

DR. BIGELOW

I think in our group, including the Sick Children s Hospital cases, in about 80 coolings there was one case of ventricular fibrillation. There have been some cardiac irregularities, and once the heart has been manipulated. We are discovering, as we investigate hypothermia more fully, that there is a dangerous side to the picture. I would like to ask Dr Lind if he could bring out one of the dangerous aspects of hypothermia. I believe he has made an interesting study

DR. LIND

The question of damage to vital structures by induced hypothermia in animals and humans has not definitely been answered. The occurrence of visceral congestion, ascites and subendocardial hemorrhages in dogs subjected to hypothermia has been reported. However, no special significance has been attached to these pathologic findings At the same time, the mechanism of hypothermic, posthypothermic and so-called "rewarming" death has not been satisfactorily explained, and systematic studies on the pathology of hypothermia seem not to have been carried out.

The material which has been studied at Nortull s Hospital by Dr H S Samuli Sarajas consisted of 24 apparently healthy dogs, unselected as to

breed, age or sex, and weighing from 5.3 to 18 5 kg. They were anesthet with intravenous Nembutal (25 mg./kg.), heparinized (2 5 mg./kg.) and tubated. Thereafter, they were cooled in an ice bath (4° to 8° C.). With exceptions, artificial respiration was used. During immersion, small amou of ether sufficient to prevent shivering were administered. For both purpo the AGA-Crafoord spiropulsator was used.

Twenty-four dogs were subjected to systemic hypothermia. Nine dogs w autopsied at the onset of fatal cardiac irregularities or at the termination moderate (26 to 27.5° C.) or deep (21 to 22.5° C.) hypothermia of 1 t hours' duration. In all cases the myocardium showed foci of necrotic mus fibers with an occasional cellular reaction. Fifteen dogs were sacrificed a autopsied 3 days to 3 years after survival of moderate or deep hypotherr of the same duration. In 13 of 15 cases distinct areas of necrosis show various stages of organization were detected.

It is evident that the myocardial alterations detected in 22 of 24 cases of pathologic nature and that the lesions in some way are related to the cool procedure. There was always a thin layer of surviving myocardium just l neath the endocardium and epicardium. This phenomenon is also charact istic of infarctions in humans and animals. Whether these lesions actually true infarctions or not remains unresolved for the present. In our dogs so stenosing processes of an apparently inflammatory nature were frequen seen in the myocardial arterioles, and the lesions were focal in nature a rather sharply delineated.

The functional significance of the lesions detected in our dogs we do know. However, it is possible that the myocardial changes described abo may lead to conduction disturbances, ectopic rhythms and myocardial insu ciency. These, in turn, may result in fatal cardiac failure particularly in t presence of other physiologic changes associated with the cooling and warming process or trauma to the heart directly, as in cardiac surgery.

DR. BIGELOW

The members of the panel might discuss myocardial irritability and so of the factors predisposing to the ventricular fibrillation which has be mentioned.

One question is rather general: Discuss myocardial irritability.

DR. EDWARDS

We have been very much interested in what effect the coronary blood flo has on the irritability of the heart. We have found in experimental anima that to exclude air emboli, one has to clamp the ascending aorta, an outflo type of occlusion.

Table 1 shows our experiences in dogs in which inflow occlusion alone w compared with outflow occlusion There was, as Dr. Scott at Vanderbi found, a much higher mortality when the outflow tract was occluded in standard ten-minute occlusion period.

We have tried to attack this problem by perfusing the coronaries and per

fusing the whole dog with arterial blood during inflow occlusion alone, to see if that would reduce the irritability of the heart.

Table 2 shows our experiences in this experiment. With no perfusion, a right ventriculotomy being the stimulus, we had 100 per cent mortality from fibrillation if we left the heart open for a period of almost ten minutes.

When we perfused the dog with 10 cc./kg./min. with a pump into the arterial system and opened the right ventricle, only 2 of 10 dogs fibrillated. When we raised that to 20 cc./kg./min. of arterial blood, we could open and close the ventricular septal defect with only one fibrillation.

TABLE 1 HYPOTHERMIA WITH INFLOW AND OUTFLOW STASIS

10 minutes

Procedure	*Number*	*Incidence of Fibrillation*	*% Mortality*
Inflow only	10	1	10%
Inflow-outflow	10	9	90%

TABLE 2 HYPOTHERMIA WITH INFLOW STASIS PLUS ARTERIAL PERFUSION (10 MIN)

Rate of Perfusion	*Number*	*Fibrillation Stimulus*	*Incidence of Fibrillation*	*% Mortal*
Control—no perfus.	10	Rt. Ventriculotomy	10	100
10 cc./kg./min.	10	Rt. Ventriculotomy	2	20
20 cc./kg./min.	10	Rt. Ventriculotomy—opening and closing septal defect	1	10

TABLE 3 HYPOTHERMIA WITH INFLOW STASIS—NO CARDIOTOMY

20 minutes

Rate of Perfusion	*Number*	*Incidence of Fibrillation*	*Permanent Survival*
10 cc./kg./min. (desc. aorta occluded)	7	2	5

Table 3 shows a series of 7 dogs in which we perfused the dogs for 20 minutes with 10 cc./kg./min. with the descending aorta occluded. Five of these dogs were permanent survivals.

We had an opportunity to try this clinically in an emergency situation which arose about three months ago in a patient with transposition. We wished to try the procedure described by Dr Albert at the Surgical Forum of the American College of Surgeons last fall, of revision of the atrial septum.

Figure 1 shows that procedure. The child was six weeks old, very cyanotic and semicomatose. We were able to occlude the vena cava and perfuse the arterial tree with arterial blood from donors The heart was occluded for a period of 19 minutes while we carried out only part of the flap revision described by Dr Albert.

The child survived the 19 minutes of occlusion with mild hypothermia

down to about 88° very well. In fact, the cyanotic heart looked a great (better when the arterial blood was being pumped in than before the occlusi The child died six hours later, apparently from cerebral anoxia, but it : been able to move all extremities after the operation.

We do feel that there is some increased irritability of the heart with red tion of coronary flow with inflow occlusion and more reduction with outf occlusion, and that there is some decreased irritability with perfusion v arterial blood.

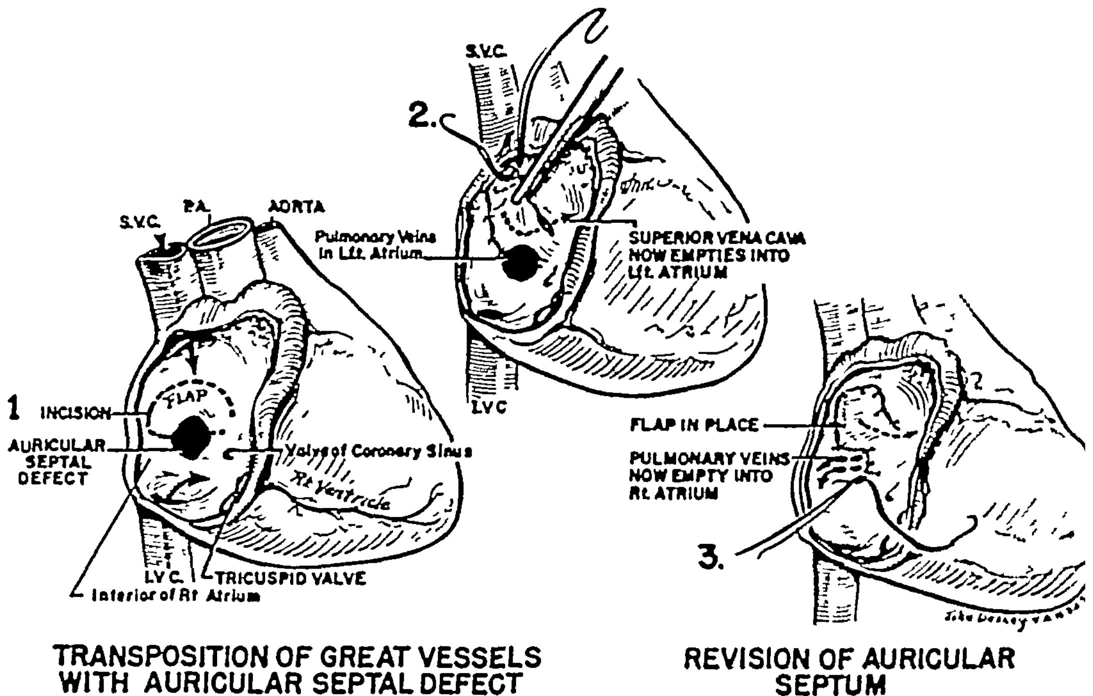


Fig 1 Diagram of operation attempted for correction of transposition of gr vessels (subject under hypothermia and with arterial perfusion with donor blood

DR. BIGELOW

Dr. Edwards, some years ago we used a two-lumen catheter and irrigat the excluded heart with oxygenated Ringer's solution as well as blood. \ certainly confirm what you have just brought to our attention.

We have carried hearts for half an hour longer, but we were always intrigu by the question of what was going to happen to the brain. What is yo thinking about that? You say you do keep the heart in a state of suspend animation for a long time. How are you going to look after the brain?

DR. EDWARDS

We are perfusing the brain in these experiments also. As I told Dr. Lillel a while ago, we are beginning to use more and more blood and less and le hypothermia.

DR BIGELOW

Dr. Dubost, · ow ´ on this vexing problem?

DR. DUBOST

I have done almost the same experiment as Dr Edwards In the experiments, perfusion of oxygenated blood at the rate of 5 cc./kg was done through a catheter by the subclavian artery with the clamping of the descending aorta. This perfusion was given during the period of vena caval occlusion, which

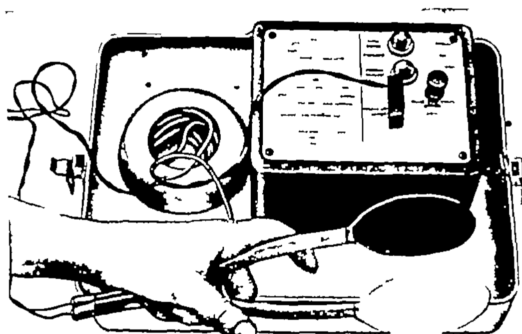


Fig. 1 Defibrillator with tong-shaped electrodes.

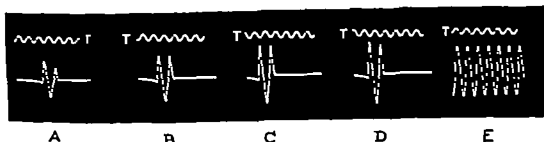


Fig. 2 Effective defibrillation current (through a fixed resistor of 50 Ohms) given by instrument of Fig. 1 *A*, 1 99 amperes. *B*, 2.37 amperes *C*, 2.82 amperes. *D* 3.21 amperes. *E*, Calibration of the 50 cycle, 127 volt standard power line through a 50 Ohms resistor 2.3 amperes. The time scale T is provided by 50 cycle alternating current. The duration of the defibrillation impulse is 0 03 sec.

lasted from 10 to 35 minutes Ventricular fibrillation occurred in 3 cases and was suppressed by adequate shock in all cases (Figs 1, 2 and 3)

In the experiments, in spite of the prolonged time of the occlusion, ventricular fibrillation was never the cause of death. When fibrillation occurred it was readily suppressed. However, all the animals in this series who were clamped for about 35 minutes, except the first one, which underwent a circulatory arrest of only 18 minutes, died within 12 hours following rewarming. Diffuse hemorrhages were observed at autopsy

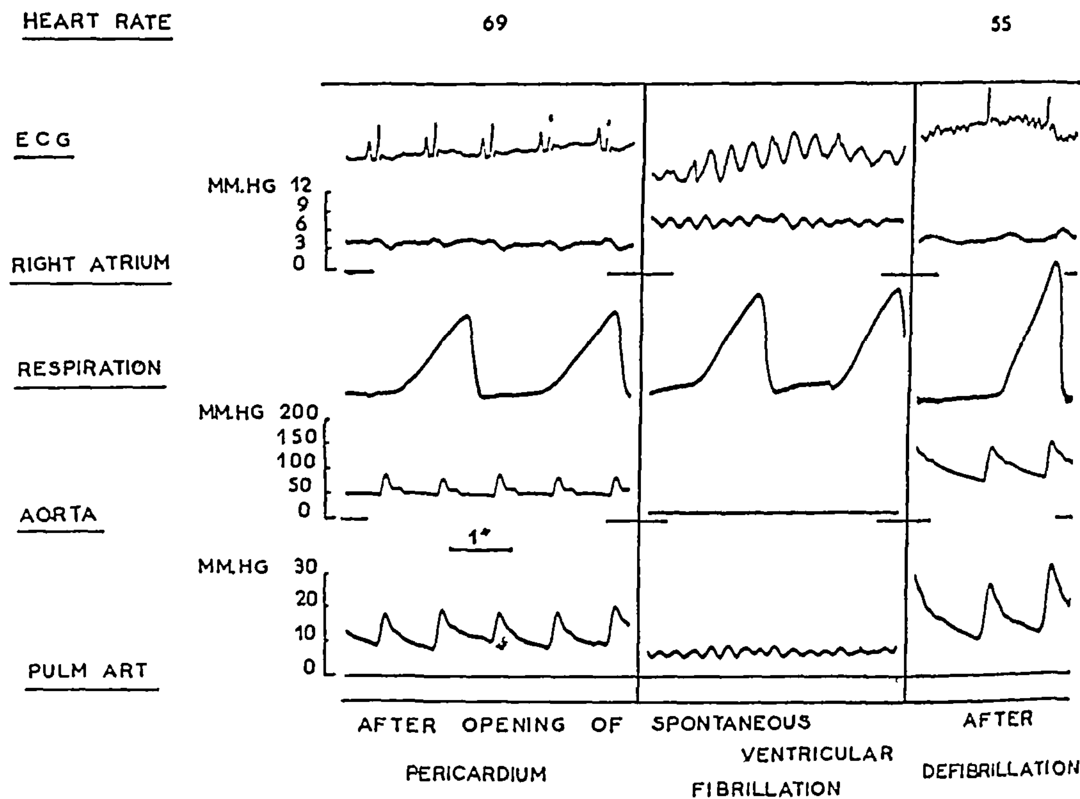


Fig 3 Spontaneous onset of ventricular fibrillation in a dog after hypothermia induced by immersion and exposure of the heart to ambient temperature (20° C) Defibrillation was obtained by a single electric shock (Fig 2, *C*) of alternating current 140 volts applied during 0 03 sec

DR. BIGELOW

Does anyone on the panel have any pertinent remarks to make on the problem of myocardial irritability to cold, and how to cure it?

DR. SHUMACKER

May I say something about its prevention? I would like to speak for just a moment about the use of sino-atrial node blockade. These experiments were performed with Dr Riberi and some of the other young men in my department.

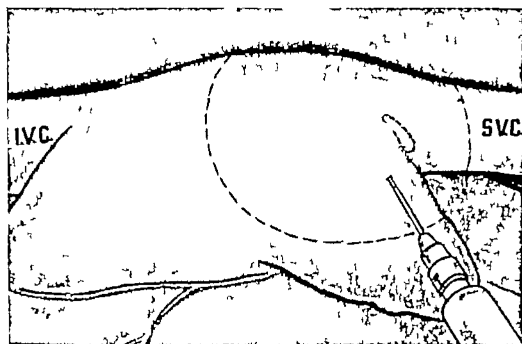


Fig. 1 Method of injection for blocking sino-atrial node The area injected is outlined with broken line.

If one clamps for 10 to 12 minutes the venae cavae of dogs cooled to from 23 5 to 28° C. and 3 minutes after the caval occlusion performs a right ventriculotomy and takes sutures in the right ventricular septum, a great difference is noted between control animals and those in which the S-A node has been blocked by procaine (Fig 1) Under these circumstances all the untreated control animals develop ventricular fibrillation and also most of those in which saline is injected instead of procaine. In contrast none of those with procaine blockade develop fibrillation and all survive (Table 1) The same striking difference is noted when other stimuli than ventriculotomy are employed.

At still lower temperatures (19 to 22° C.) 53 per cent of untreated animals develop fibrillation during right ventriculotomy, whereas this occurs in only one animal out of 15 (6.5 per cent) protected by sino-atrial node blockade.

The injection of procaine in the region of the S-A node brings about a remarkable slowing of the heart. The P waves disappear or become strikingly altered (Fig 2)

TABLE 1 SINO-ATRIAL BLOCKADE AND VENTRICULAR FIBRILLATION

Temperature	*Treatment*	*Number of Animals*	*Percentage Developing Fibrillation*
	External Stimuli and Digital Septal Massage		
	None	6	100
23.5–27.5	Saline Injection	2	100
	Procaine Block	8	0
	Right Ventriculotomy and Suture of Septum		
	None	6	100
24.6–28	Saline Injection	6	67
	Procaine Block	12	0
	None	15	53
19–22	Procaine Block	15	6.5

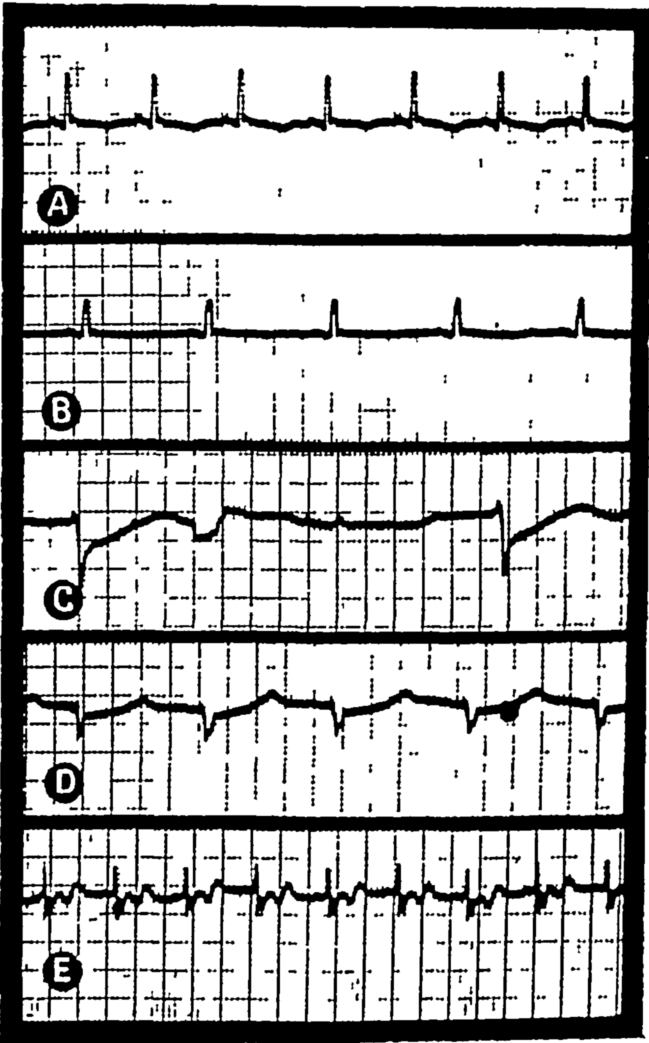


Fig. 2. Electrocardiographic tracings of dog subjected to S-A procaine blockade, right ventriculotomy, and suture of nylon plate to ventricular septum. *A*, Before injection. *B*, After injection. *C*, Circulation restored. *D*, Pericardium closed. *E*, Following day.

This method is remarkably effective. Dr Radigan and Dr Morrow have repeated this work at Bethesda and have confirmed it. Professor Lian's group in Paris has been interested in the same problem.

As Dr Riberi mentioned in discussing Dr Lillehei's presentation, we have been able to make and close ventricular septal defects in dogs without any mortality whatsoever utilizing this protective maneuver I do not mean to infer that because one can do ventriculotomy safely upon the normal hearts of dogs, it necessarily follows that the same is true with the disordered hearts of patients. I may say that I have operated upon one extremely ill patient with a large ventricular defect under hypothermia and with the S-A node blocked. The heart was opened, the ventricular septal defect was repaired, and the patient regained consciousness and good circulation. For some reason that I do not understand the patient died late the night of operation after having had some twitchings in one extremity I presume that we may have had air embolism to the cerebral vessels

The use of this principle does hold real promise for reducing the incidence of ventricular fibrillation during cardiac procedures carried out in the hypothermic state.

DISCUSSION

Henry Laborit (*Paris*)

FIRST PROBLEM. HOW TO ARRIVE AT THE FUNCTIONAL THERMIC OPTIMUM

This factor is related to the ratio $\frac{Ca}{K}$[1] The optimum functional temperature drops with the decrease in potassium serum level and the increase in calcium serum level. *This phenomenon applies to the whole body In the state of hibernation*, the true hibernating animal is in hypokalemia (from pancreatic activity, potassium enters the cells and potassium serum level is lower) Parathyroid glands still function and maintain a hypercalcemia.[2] It has also been found that in normal sleep there is an increase of the ratio $\frac{Ca}{K}$

In fact hypokalemia in itself is not sufficient; there must be a hyperkalicytia, that is an increase of cellular potassium which corresponds to an increase of the normal membrane electric potential. This fact has been brought out by the study in skeletal muscle of the excitability curve obtained with several values of excitation intensity and length of excitation.[3]

(1) How to obtain hyperkalicytia with hypokalemia.

(a) Use of lytic drugs plus insulin and glucose.[4]

(b) Hyperventilation which causes hypocapnic alkalosis (Swan) Neostigmine gives the same result.

However, in our opinion, the Swan technique only prevents potassium from passing outside the cell, which is what occurs normally with sudden refrigeration under normal anesthesia, this state of hyperkalemia has been demonstrated by Bigelow[5] and Juvenelle et al. In our opinion there is danger

in the alveolar hyperpressure as caused by assisted respiration because it increases the vascular pulmonary resistance.

(2) How to obtain hypercalcemia.

(a) By intravenous injection of calcium salts (however, calcemia does not increase much, the same applies to the urine-calcium level), at 37° C. calcium renders cell membranes impermeable.

(b) On the other hand, in hypothermia, calcium lowers the melting point of membrane lipids (Monnier)[6] and maintains permeability of cell membranes which otherwise would have been diminished by refrigeration. The metabolism is, therefore, less depressed but the functional optimum is maintained and the metabolism is strictly regulated according to van't Hoff's law. Kayser[7] has shown that at 25° C. the metabolism of the truly hibernating animal is higher than when the same animal is artificially refrigerated to the same temperature and under anesthesia.

With hypercalcemia it is, therefore, possible to obtain a deeper hypothermia (19–20° C) and at the same time to keep spontaneous respiration. The systolic cardiac tonus remains normal because of the maintenance of an efficient activity electric potential. Hyperkalicytia is accompanied by a decrease of the basic tonus, these two factors result in a better cardiac efficiency and in an increase of the differential pressure.

However, at the same temperature the cardiac rhythm is increased and in order to benefit from a lower metabolism, a deeper hypothermia is necessary and this follows van't Hoff's law.

Furthermore, a good intracardiac conductivity remains at temperatures of 19 to 20° C, since calcium maintains the permeability of the membranes in hypothermia

Calcium in hypothermia has appeared to us as being the perfect adjunct to neuroplegics and refrigeration because it maintains the activity current of the heart without altering the rest potential.

SECOND PROBLEM. UNDER CLAMPING, THAT IS ANOXIA, THE HEART STILL HAS TO MAINTAIN A CERTAIN ACTIVITY.

Bing's work has shown that under clamping the cardiac fiber consumes no lactic acid or ketones but does consume carbohydrates, therefore, it is of interest to increase the glycogen content of the cardiac fiber.

Two years ago we found experimentally that the somatotrophic hormone decreases significantly the incidence of fibrillation in hypothermia.[8] Recently Illingworth has shown that the same hormone increases considerably the glycogen content of the myocardiac fiber.

It is also useful to supply at time of declamping the energy-supplying material which is necessary to the restoration of the myocardiac tissues. This is obtained with oxalo-acetic acid, glutamic acid, alanic acid or aspartic acid, which are immediately consumed in Krebs' cycle[9], cytochrome or ATP may also be of interest

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ANEURYSMS AND OCCLUSIVE DISEASES OF THE AORTA

MICHAEL DEBAKEY (*Houston*)—CHAIRMAN

SURGICAL TREATMENT OF THORACIC ANEURYSMS

HENRY T. BAHNSON (*Baltimore*)

This presentation is limited, like many surgeons, by the diaphragm. The diaphragm is a good boundary because thoracic aneurysms differ in etiology, clinical characteristics and method of handling from those in the more distal aorta below the diaphragm.

ETIOLOGY

The reason for this division becomes more apparent when we consider the two main causes for aneurysms of the aorta, syphilis and arteriosclerosis. Syphilitic aneurysms are much more apt to be saccular, occur in the thoracic

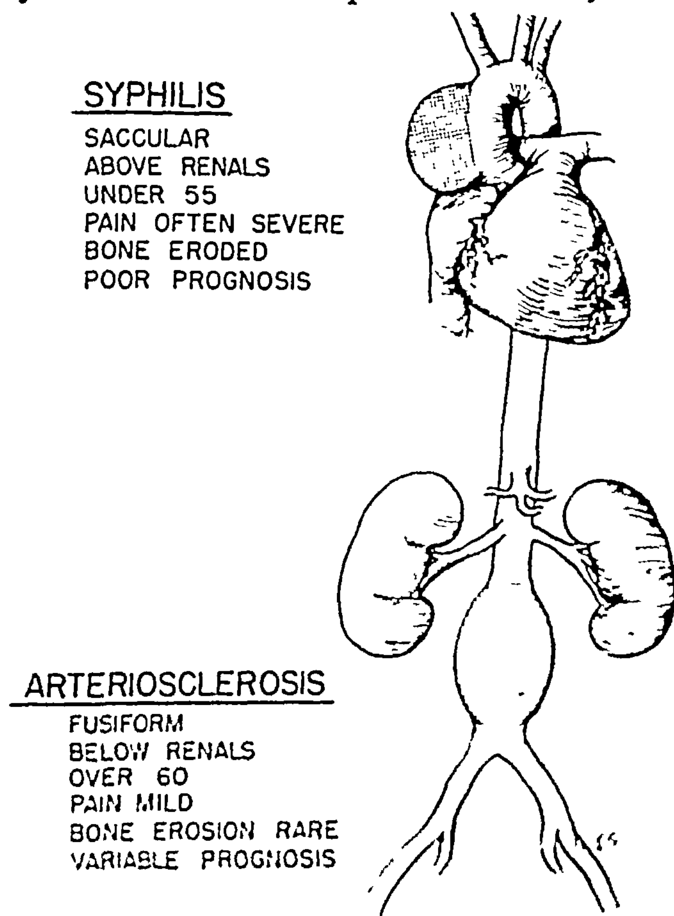


Fig 1. Chart of location and clinical characteristics of the two principal types of aortic aneurysms. Occasional aneurysms due to trauma or bacterial infection resemble those due to syphilis in most respects (From Bahnson: Ann. Surg., vol 138, 1953)

or upper abdominal aorta, in middle-aged individuals and more often produce symptoms and signs than arteriosclerotic aneurysms, which usually are fusiform, occur below the renal arteries, in older patients and without distressing symptoms (Fig. 1) This distinction is not invariable, however, as there are occasional aneurysms in the thoracic aorta which are caused by arteriosclerosis, some of which are saccular, and there are abdominal aneurysms caused by syphilis.

There are additional causes for uncommon aneurysms. Automobile accidents associated with a sudden stop and a shearing force exerted on the thoracic aorta may cause a tear of the aorta with sudden death, or occasionally the tear is small enough so that it is contained by adjacent tissue and sealed by clot. This may liquefy and form an aneurysm. There have been several aneurysms reported which were considered congenital in nature because of the virginal appearance of the aorta and aneurysm and the presence of other congenital anomalies In connection with congenital aneurysms, a right aortic arch should be mentioned because of two examples recently brought to my attention which were thought to be saccular aneurysms of the aorta, until it became clear that the apparent aneurysm was due to a fusiform dilatation of a right aortic arch. In some instances, such a dilatation of a right arch may be associated with tracheal or esophageal obstruction, perhaps aggravated by an aortic ring Infection with organisms other than the Treponema pallidum may cause aneurysms. These, those due to trauma, and the uncommon true congenital aneurysm in most clinical respects simulate syphilitic aneurysms To be complete, one must include coarctation of the aorta, which is frequently associated with a post-stenotic aneurysmal dilatation just distal to the coarctation. This, on some occasions, may assume aneurysmal proportions Of greater clinical significance are aneurysms of the intercostal arteries which develop as a result of the increase of collateral flow around the coarctation

PROGNOSIS

Thoracic aneurysms and more particularly those due to syphilis, concerning which better statistics are available, are associated with a generally poor prognosis. Only 18 of Kampmaier's[1] 633 patients lived for more than two years following the first symptoms Most of them died within the first six to nine months This amounts to a 3 per cent two-year survival. In addition to the poor prognosis, these aneurysms invite surgical attack because of pain from pressure on spinal nerves or obstruction of the esophagus or tracheal-bronchial tree.

TREATMENT

Many methods of treatment have been advocated for thoracic aneurysms but only three merit consideration in this brief presentation. Fibrogenic materials, popularly used as cellophane, have been frequently employed in the past. The apparent simplicity of its use is certainly one reason for this However, on the basis of treating 32 patients Abbott[2] felt that life was not prolonged in any of his patients with advanced disease, and the only benefit claimed was relief of pain in 40 per cent of the cases In an effort to induce

thrombosis of the aneurysm, several types of wire have been inserted into aneurysms. The greatest refinement of this technique is that of Blakemore and King[3] with fine coin silver wire which can be heated following insertion. Blakemore's[4] ten-year follow-up on patients with saccular aneurysms treated by this technique showed that only 27 per cent were living, although most of these were symptom-free and working.

EXCISION. The most appealing form of treatment, and the one which we are employing exclusively at the present time, is that of excision. This is not a new technique as Tuffier,[5] in 1902, demonstrated that most syphilitic



Fig 2. Aortogram showing opacification of aneurysm by contrast medium injected through a catheter inserted percutaneously in a femoral artery. (From Bahnson: Ann. Surg, vol 138, 1953)

aneurysms have a relatively narrow mouth which can be isolated and occluded with subsequent closure of the aortic opening. Tuffier's efforts met with failure largely because of infection.[6]

The treatment of coarctation of the aorta by Crafoord, Gross, and Blalock and Park and the impetus given to vascular surgery by the contributions of Blalock and Taussig were responsible for the development of techniques and thinking which made excision of aneurysms the method of choice. Indeed the first aortic aneurysms which were excised were in association with coarctation of the aorta.[7] The presence of coarctation facilitates the operation in that the aorta may be occluded for a longer time because of the extensive collateral circulation. By the same token, however, the collateral circulation and enlarged intercostal arteries may make bleeding a more serious problem.

In the last ten years several reports have appeared of success in the excision

of aortic aneurysms, although most authors did not advocate excision for saccular syphilitic aneurysms Blakemore,[4] in 1947, and Cooley and DeBakey,[8] in 1952, reported successful excision of aneurysms of the innominate artery involving the aorta, and the latter authors advocated excision for intrathoracic aneurysms of the aorta and great vessels Subsequent work has shown that aneurysms in any portion of the aorta or great vessels are amenable to excision and that this is the method of choice.[6 9] This is possible because, although syphilis may involve the entire cardiovascular system, most syphilitic aneurysms represent localized blow-outs If this localized area is excised, the

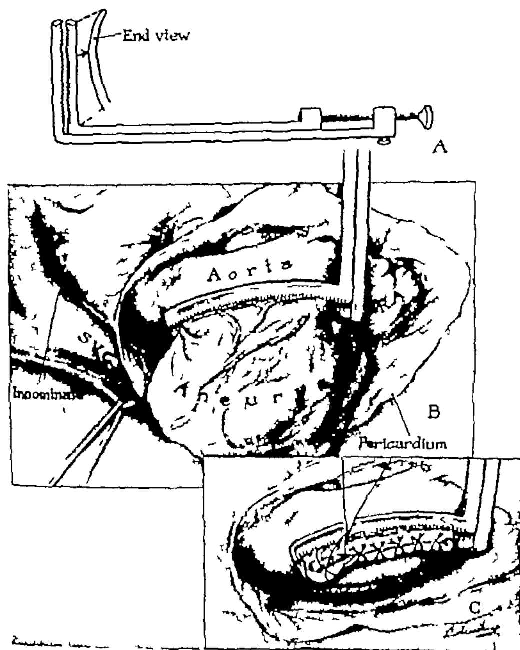


Fig. 3 Clamp devised for occlusion of mouth of aneurysm. Operative exposure of aneurysm on the ascending aorta through third right interspace and vertically split sternum. The mouth of the aneurysm has been dissected free and the clamp applied. After excision the mouth was closed with multiple interrupted sutures of 0000 silk.

remaining portion of the cardiovascular system may be adequate for years of satisfactory service. Traumatic, mycotic and congenital aneurysms are usually even more localized.

Several areas of the *thoracic* aorta present individual problems in management. Figure 2 shows an aneurysm on the ascending aorta, a frequent site of syphilitic aneurysms, which has been opacified by contrast medium injected

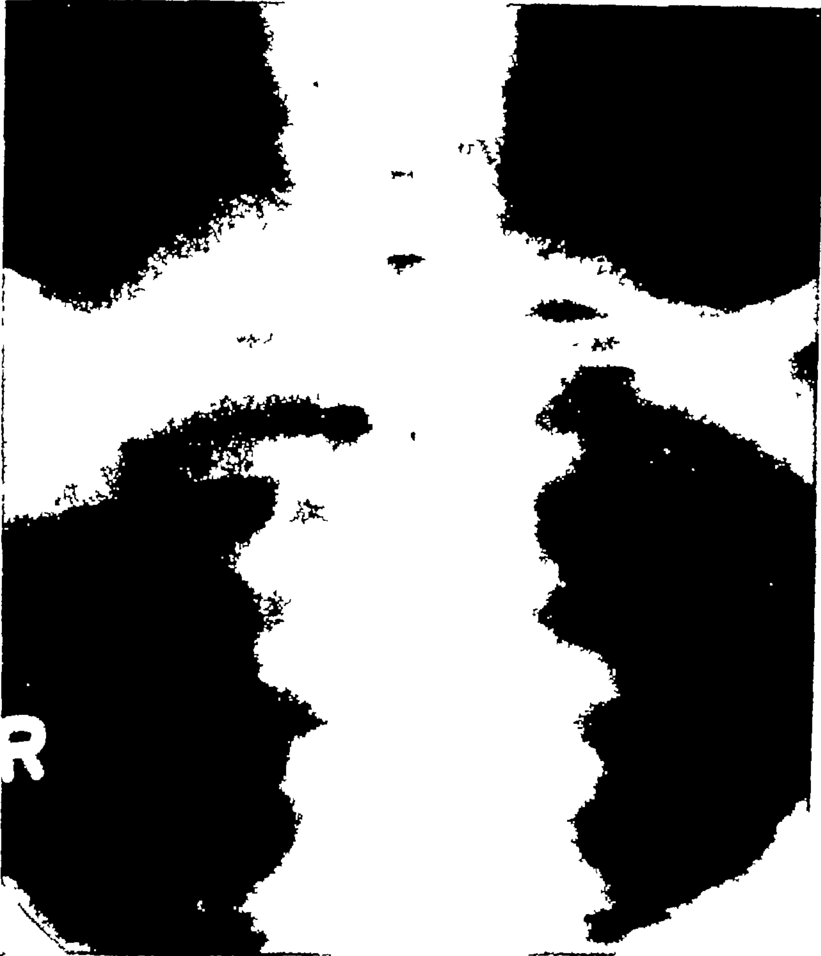


Fig 4 Laminogram of small but embarrassingly located aneurysm of the aortic arch The patient was dyspneic at rest. (From Bahnson. Surg, Gynec. & Obst, vol 96, 1953)

through a catheter inserted percutaneously into a femoral artery.[10] Aneurysms in this location must be sufficiently saccular, which fortunately is usually the case, to allow isolation of the neck and clamping of relatively normal adjacent aorta. Any of several instruments may be used to occlude the mouth of such aneurysms. We have found most satisfactory a heavy instrument with multiple teeth which is occluded by a screw mechanism rather than the customary scissors action (Fig. 3). Closure of this and other aneurysms has been performed by multiple sutures of 0000 silk. We have found it difficult to use curved needles in the tough tissue and usually employ shortened Halsted intestinal needles.

It is imperative in treating such patients that dissection first be carried out in areas which are relatively uninvolved, and only after the adjacent aorta and mouth of the aneurysm have been isolated should dissection about the aneu-

rysm itself be performed. In such instances boldness may be required, but it is important that such boldness be preceded by careful dissection wherever possible.

The *innominate artery* is the second most common site of intrathoracic aneurysms and these may be handled in a similar fashion. The aorta is usually free, and the subclavian and carotid artery may be isolated. In all 5 patients

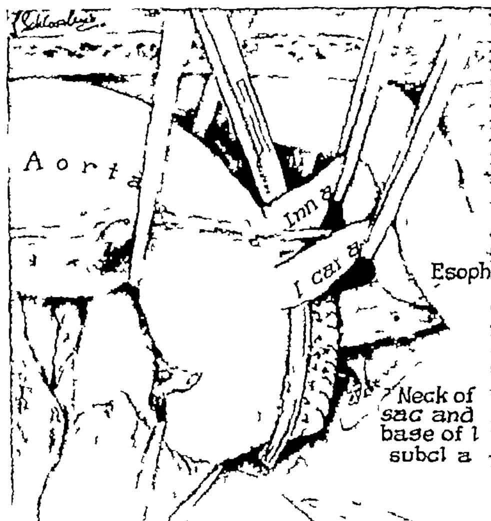


Fig. 5 Operative exposure through the second left interspace and split sternum Aneurysm and left subclavian artery were excised after occlusion of the mouth of the aneurysm on the convexity of the aortic arch by a coarctation clamp (From Bahnson, Surg., Gynec. & Obst., vol. 96 1953)

we have treated with innominate aneurysms we have simply excised the aneurysm without attempting to reconstruct the connection between the carotid and the aorta. We have been hesitant to attempt reconstruction of continuity for fear of small cerebral emboli and also because of the greater difficulty of performing an anastomosis with the thick tissue of the aneurysm mouth than of closing the opening It is usually possible to leave the subclavian and carotid in communication. In none of the cases has ligation of the innominate caused a noticeable detriment to the cerebral circulation.

Some of these aneurysms may be relatively small and yet associated with



Fig 6 Aortogram and superimposed specimen from patient with aneurysm of th aortic arch

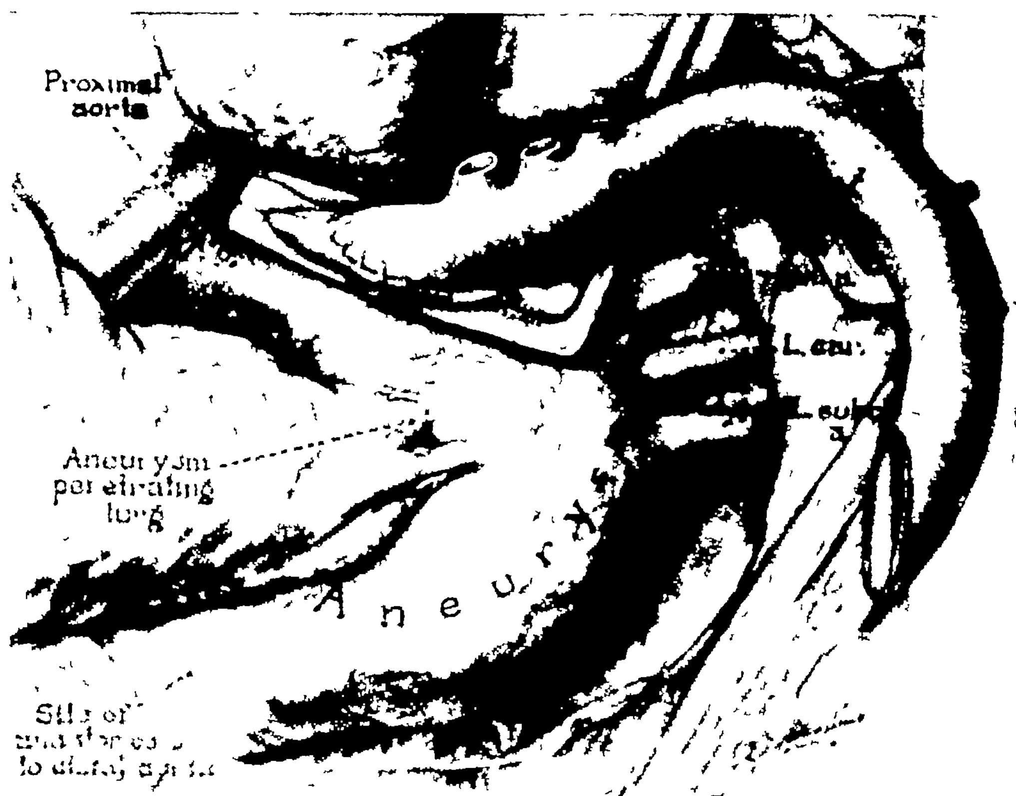


Fig. 7 Operative exposure through the left fourth intercostal space and vertically split sternum A shunt graft has been sutured to the side of the ascending aorta and was completed to the distal aorta before removal of the aneurysm.

striking embarrassment to the patient. Figure 4 shows the laminogram of a small aneurysm on the aortic arch which was responsible for considerable dyspnea even at rest. It was readily isolated and excised (Fig 5), and the patient when last contacted was well, although in the Maryland House of Correction.

In order to excise the aortic arch itself one must have some means of maintaining flow to the body and, equally or more important, out of the heart. For this purpose we have used a shunt graft as first described by

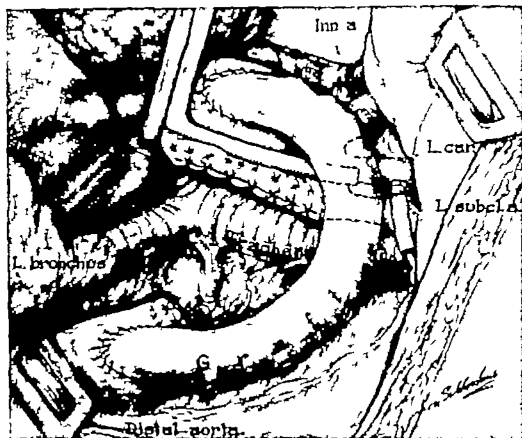


Fig 8. Completed shunt graft after excision of the aneurysm. A tongue of the greater curvature of the arch could be preserved with the great vessels arising from it. Excessive length of the graft was required to circumvent the adherent lung.

Mahorner and Spencer [11] This can be sutured into the ascending aorta and consecutively to the innominate, carotid, subclavian and distal aorta. In Fig 7 is shown such a graft in place on the ascending aorta. We thought from the aortogram (Fig 6) that the more proximal portion of the arch was involved and had planned to anastomose the vessels of the arch to the graft. We were able to save a tongue of the greater curvature, however, and left the vessels attached to the patient's arch. The anastomosis to the distal aorta was completed before the aneurysm was excised (Fig 8) It is important whenever possible to have *circulation so maintained*, as *mobilization of such aneurysms* cannot safely be done without occlusion of the aorta and may be tedious and time-consuming because of adherence to pulmonary artery, bronchus or penetration into the lung An excessively long graft may be required, as in this



Fig. 9 Posterolateral exposure of aortic arch and mediastinum The aneurysm arose from the distal portion of the aortic arch The aneurysm was opened and excised after the aorta was clamped proximally and distally. The circumferential tear in the aorta was sutured and adjacent aneurysm sac sutured over the defect (From Bahnson Surg , Gynec & Obst , vol 96, 1953)

case, to get around the aneurysm and lung, but the patient has done well, and the extra vessel seems to be no problem.

In the more distal portions of the arch the aorta may be cross-clamped as shown in Fig. 9 This patient had an aneurysm which resulted from an automobile accident. This is a common site for nonfatal injury to the aorta, just distal to or at the origin of the left subclavian artery. In this individual the aorta was occluded between the innominate and carotid arteries and just distal to the aneurysm for 7 minutes while the aneurysm was incised and

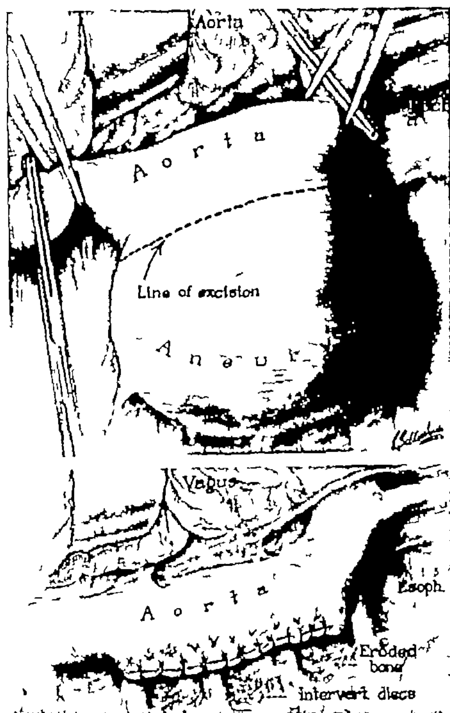


Fig. 10 Aneurysm of upper thoracic aorta treated by lateral aortorrhaphy during aortic occlusion.

closure of the mouth was begun. Seven minutes must have been near the limit of tolerance in this individual for at the end of this time her pressure began to fall and frequent extrasystoles appeared. A portion of the aneurysm was held against the remaining opening as the clamps were removed and further sutures placed to complete the closure.

We have treated aneurysms of the thoracic aorta in one of two ways In most instances these aneurysms have been saccular The aorta above and below the aneurysm may be occluded, the sac incised and the aorta recon-

structed from adjacent aortic wall (Fig 10). Usually one may clearly see a line of demarkation between aorta and aneurysm.

This is perhaps a fitting time to comment upon the important role which antibiotics play in the treatment of these patients, for this patient appeared with both the aneurysm and a large tuberculous cavity in his right upper lobe. The aneurysm appeared to be of prime importance, and this turned out to be correct, as after excision of the aneurysm he was given six months of antituberculous therapy, a residual cavity was removed, and he is now well

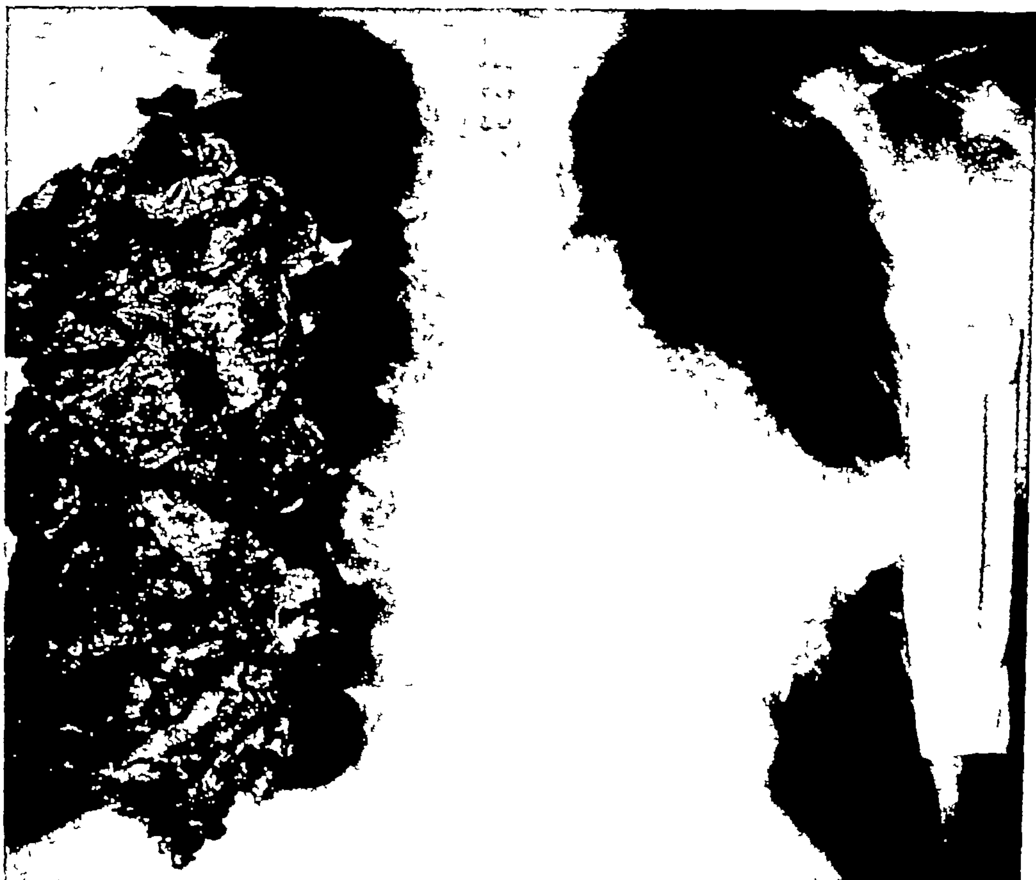


Fig 11 Roentgenogram of patients with aneurysm in midthoracic aorta with superimposed specimen and nylon graft

I am sure that the antibiotics cover up many of our surgical imperfections and the patients' susceptibilities

We had hoped to treat the patient shown in Fig. 11 by a similar procedure but on opening the aneurysm found no line of demarkation. We did not have a homograft prepared and during 24 minutes of aortic occlusion inserted a tube of nylon fabric similar to that shown.

Means have been described to prolong the period of safe occlusion for excision of aneurysms of the aortic arch and beyond. Mahorner and Spencer[11] have sutured a by-pass homograft in place prior to excision of an innominate aneurysm. Stranahan and associates[12] described a similar approach and advocated the use of frozen-dried steer vessels as a temporary shunt while the normal aortic channel is being reconstructed. The use of temporary shunts has been advocated and used by some [13,14] The only patient we have seen

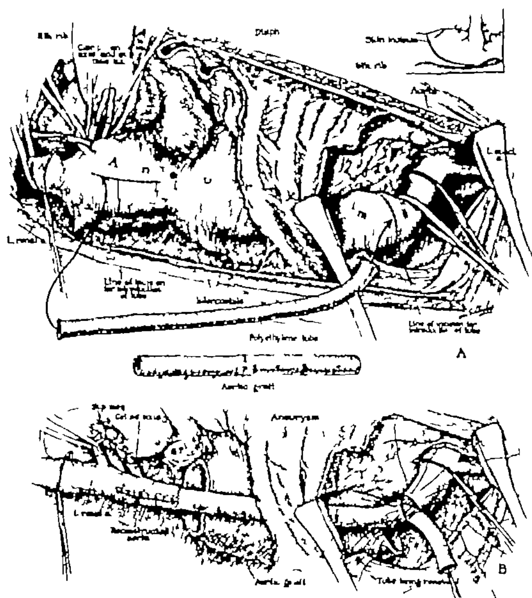


Fig. 12. Extensive arteriosclerotic aneurysm involving the aorta from the left subclavian to renal arteries. Aorta was reconstructed with a homograft around the intraluminal polyethylene shunt. The shunt was removed on completion of the upper suture line. The upper abdominal aorta was reconstructed by an aneurysmorrhaphy prior to insertion of the homograft.

in whom such a shunt seemed necessary had an aneurysm which extended from the subclavian to the renal arteries The aorta was reconstructed by an aneurysmorraphy from the lower end to just above the celiac axis, and the graft was sutured in place over a shunt from this level to the upper thoracic aorta (Fig 12) This technique is similar to that used by Lam and Aram in the Henry Ford Hospital in 1950 Our patient was paraplegic following operation and failed to recover from the massive surgical procedure. It is probable that division of intercostal arteries over this wide an area may be responsible for some damage to the spinal cord.

Finally, hypothermia has been advocated by Pontius and associates[15] as a means of obtaining a longer safe period of aortic occlusion. Occlusion of the upper thoracic aorta of over an hour without evident residuum has been described. To date we have been fearful of hypothermia, and in the thoracic aorta I prefer the risk of aortic occlusion without the additional hazard of cooling. About the arch I prefer a shunt graft. One patient, in addition to the one shown in Fig. 12, has suffered weakness to the lower extremities as evidence of cord damage from interruption of the circulation. In this individual a forgotten clamp was inadvertently left on the aorta for a total of 45 minutes, 7 minutes longer than the time required to complete the anastomoses.

Several points in general technique might be mentioned. A fatal infection three months after the first successful excision of a thoracic aneurysm and grafting which Lam and Aram performed in this hospital, and the patients of Tuffier, and of Cooley and DeBakey, offer sufficient evidence that the sac and contents must be removed as completely as possible to prevent later infection and possible rupture of the suture line. We have had 3 patients return with infection in the region from which the aneurysm was excised. In all 3 the aorta reopened At the initial operation in the first patient it was thought that both innominate and left common carotid arteries were involved in the aneurysm and that both had inadvertently been divided. Bleeding was controlled with difficulty and the aneurysm only partially excised. After operation it was apparent that only the innominate had been divided. The aneurysm reformed and became infected with a hemolytic Staphylococcus albus which was resistant to available antibiotics except erythromycin and did not respond to this. At reexploration six months after the initial procedure the leaking aortic opening was refashioned, sutured with steel wire and the remaining aneurysm cleanly removed. The patient has since done well for almost a year. A similar attempt ended unsuccessfully in the second case, and the third died of a massive mediastinal abscess. In neither of the latter two patients were we aware of leaving a particularly untidy wound. Except for these 3 cases there has been no evidence of late trouble from either operation or aneurysm in this group of patients.

Exposure of the area must be liberal. When the anesthetist has proper control of respiration one need have little fear of splitting the sternum or of entering the opposite pleural cavity. For aneurysms on the ascending aorta or arch we have used an incision through the second, third or fourth interspace, and the sternum has been split upwards in the midline or transected to the opposite side Aneurysms of the thoracic aorta have been treated by a large standard posterolateral incision.

Patients who are dyspneic because of aneurysms of the aortic arch are characteristically least dyspneic when leaning forward. One patient seen by the author, in whom incision was planned, died of anoxia following bronchoscopy. An additional patient convulsed twice from anoxia while lying supine during radiographic examination. Hyperextension, or even extension, may close off the trachea and at the same time increase the demand for oxygen because of excitement. The luxury of special studies may have to be forfeited in such individuals.

Care must be taken to avoid dislodgment of clot from the aneurysm to the distal circulation. This is particularly true with aneurysms on the ascending aorta. During mobilization of such aneurysms and application of clamps, the carotid arteries should be occluded intermittently in the neck. One patient died as a result of dislodging a calcified clot from the rim of the aneurysm to the right internal carotid artery, and in an additional patient embolus was successfully removed from the aortic bifurcation.

Since February 1952 the residents of the Johns Hopkins Hospital and I have attempted excision of 26 thoracic aneurysms. This does not include several aneurysms of intercostal arteries or of the aorta in association with coarctation of the aorta. There have been 8 deaths, all the remaining patients are well and symptom-free.

In conclusion, in 1902 Tuffier stated that for aneurysms of the aorta to be amenable to surgery, they should be "accessible, isolable and extricable." Surgical progress in the past fifty years has allowed us to catch up with Tuffier's foresight.

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ANEURYSMS OF THE ABDOMINAL AORTA: ANALYSIS OF 101 CASES TREATED BY EXCISION

Denton A. Cooley, Michael E. DeBakey, and
Oscar Creech, Jr. (*Houston*)

Aneurysm of the abdominal aorta is a serious disease producing in many instances disabling symptoms and ultimately leading to death from rupture In the past, methods of therapy were directed toward obliteration of the aneurysm by inducing thrombosis within the lesion or by reinforcing the wall to forestall perforation Such procedures proved to be inadequate and provided temporary relief of symptoms in relatively few instances In our series of cases, for example, there were 8 in which the aneurysm had been previously wrapped with a polyethylene film coated with dicetyl phosphate, a tissue irritant which promotes fibrosis Although the aneurysms had been wrapped for varying periods up to 26 months prior to excision, the degree of fibrosis about the wall of the lesion was slight and progressive enlargement of the aneurysm had occurred in all instances. Indeed, it appeared that the growth of the aneurysms was enhanced by the presence of the film.

Introduction of wire into the aneurysm was a method of treatment designed to promote thrombus formation in order to reinforce the wall or actually obliterate the aneurysm. For the most part results of this method of therapy even in combination with partial ligation of the parent vessel were unsatisfactory This is illustrated by two cases in our series of resection in which progressive enlargement followed the wiring procedure In one of these patients there was rapid progression of symptoms and striking enlargement of the aneurysm during the 7-month interval between the wiring procedure and the resection (Fig 1). A roentgenogram of the abdomen revealed that a large pack of wire had been introduced into the lesion and some of the wire extended proximal to the visceral branches of the abdominal aorta (Fig. 2). Resection of the aneurysm with the contained wire pack was accomplished, and aortic continuity was reestablished by means of an aortic homograft (Fig. 3). On examination of the specimen it was evident that the small, soft thrombus produced by the wire pack was an ineffective means of controlling the disease process (Fig. 4).

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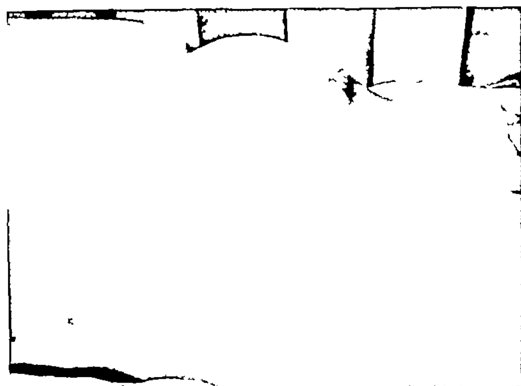
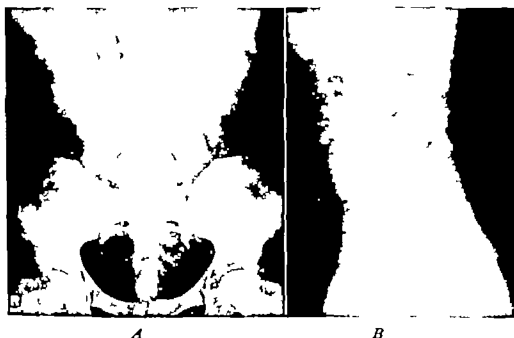


Fig. 1 Photograph of abdomen in side view of patient 7 months after wiring procedure of abdominal aortic aneurysm shows the striking silhouette of the large pulsatile mass which was quite painful.



A B

Fig. 2. Roentgenograms of abdomen in same patient as in Fig. 1 demonstrating in the anteroposterior (*A*) and lateral views (*B*) a wire pack in the aneurysm and extending proximally into the aorta itself.

During the past few years excisional therapy of aortic aneurysms with graft replacement has become the method of choice in all lesions of the abdominal aorta in which conditions permit its satisfactory application. The



Fig 3 Photograph made at operation on the patient in Fig 1 after excision of the aneurysm and replacement with a lyophilized segment of aortic bifurcation



Fig 4. Photograph of specimen of aneurysm showing wire pack in aneurysm and demonstrating a relatively small degree of associated thrombosis The segment of wire shown above was removed from the proximal aorta

purpose of this report is to present an analysis of 101 cases of abdominal aneurysm treated in this manner.

Aneurysms of the abdominal aorta are usually arteriosclerotic in origin and fusiform in type. Characteristically the lesions occur in that segment of aorta distal to the origin of the renal arteries and extend distally to involve the

aortic bifurcation and frequently the iliac arteries Thus, in our consecutive series of 102 patients operated upon for abdominal aneurysm, in only one case did the lesion extend above the level of the renal arteries and other major visceral branches of the aorta, and this lesion was probably caused by syphilis. Accordingly, in the entire series this was the only patient in whom excisional treatment was not attempted. The uniform location of aneurysms of this type below the renal arteries is of considerable clinical significance since the finding of a pulsatile abdominal mass indicates that an operable aneurysm exists. For this reason, too, diagnostic lumbar aortography has become an unnecessary procedure in determining operability or in planning the surgical approach. Selection of patients for operation in this series depended solely upon the diagnosis of aneurysm, and no patients were denied operation even though many were aged and had serious cardiovascular, renal or respiratory disease which increased the risk of operation. The belief that the aneurysm itself provides the greater risk is supported by our experience with one patient who while awaiting operation for several days because of a bladder disturbance died suddenly from rupture of the aneurysm with exsanguinating hemorrhage.

TECHNIQUE

Laparotomy is performed through a midline incision extending from several centimeters below the xiphoid process to a midpoint between umbilicus and pubis The small intestine is usually delivered from the peritoneal cavity and retracted toward the right using warm moist packs The peritoneum over the root of the mesentery is incised, and the aneurysm is exposed. Proximal to the aneurysm mobilization of the aorta is done, carefully avoiding damage to the left renal vein, and the aorta is encircled with an umbilical tape just below the origin of the renal arteries The iliac arteries distal to the aneurysm are similarly mobilized, avoiding damage to the underlying iliac veins In all cases the inferior mesenteric artery has been divided and none have shown evidence of ischemic changes to the left colon or rectum.

Preparations are made for use of a lyophilized or freeze-dried aortic homograft or an orlon or nylon woven or knitted cloth prosthesis to bridge the defect in the aorta created by the resection. Selection of the type of graft or prosthesis and the advantages of each are presented in another section of this symposium. Upon completion of these preparations, the aorta is occluded proximally with a minimum trauma clamp Distally the iliac arteries are occluded with bulldog clamps, and the aneurysm is excised usually from below upward, ligating paired lumbar arteries posteriorly In most instances the aneurysm is adherent to the inferior vena cava and in some instances a small plaque of aneurysmal wall is left attached to the vena cava. Division of the aorta proximally provides a short cuff of vessel sufficiently long to permit satisfactory suture to the graft. The proximal aortic anastomosis is performed using 4-0 arterial silk and the distal iliac anastomoses with 5-0 silk. Usually the proximal aortic anastomosis and one iliac anastomosis are done first and circulation is restored through this channel as the other iliac anastomosis is completed. Thus total interruption of aortic flow seldom exceeds 45 minutes,

and the remaining iliac anastomosis may be completed in an additional 10 to 15 minutes. As circulation through the graft is commenced, moderate bleeding occurs from needle holes but is easily controlled by gentle pressure. The peritoneum is sutured over the graft and the abdominal incision is closed without drainage.

In most cases of abdominal aneurysm the disease process involves the aortic bifurcation, and frequently aneurysms of the common iliac arteries are present. Therefore it becomes necessary to replace the aortic bifurcation in the majority of cases, as was done, for example, in 92 of 101 aneurysms in our series (Fig. 5). Thus, in only 9 instances could the aortic bifurcation be satis-

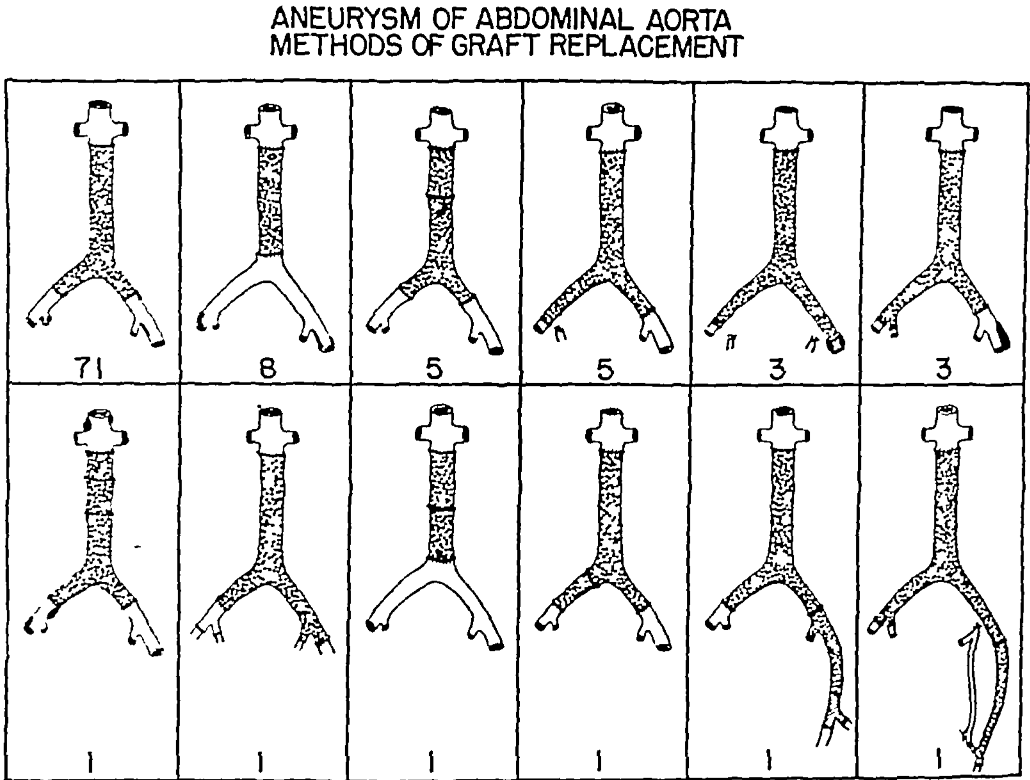


Fig 5 Drawing showing various methods of restoration of circulatory continuity employed in 101 cases after resection of arteriosclerotic abdominal aneurysm, and the number of patients in which each was used

factorily preserved. A number of variations in the grafting procedure have been employed because of local factors which may require improvisation (Fig. 5). For example, if the diameter of the proximal aorta is large it may be necessary to utilize a segment of thoracic aortic graft for the proximal anastomosis, and this graft is sutured to the bifurcation graft of abdominal aorta distally. In many instances these technical problems are solved by tailoring the end of the graft to produce a greater circumference. It is of prime importance in all instances to restore a pulsatile flow into the vessels distally and provide as normal circulation as possible in the presence of extensive arteriosclerosis

In addition to the aortic resection done in these 101 cases, other abdominal procedures have been performed at the time of laparotomy (Table 1). For

example, in every patient in whom appendectomy has not been performed at previous operation, the appendix is removed, as was done in more than half of the cases Bilateral lumbar sympathectomy was performed routinely in the first 25 cases, but at present it is only done in cases in which there is evidence of severe peripheral vascular occlusive disease. Cholecystectomy was done in 8 patients, and in 1 patient who was deeply jaundiced a number of large common duct stones were removed. In another patient gastric resection was done because of the presence of a stenosing duodenal ulcer The fact that

TABLE 1 ABDOMINAL ANEURYSMS ASSOCIATED SURGICAL PROCEDURES (101 CASES)

Appendectomy	More than half the cases
Cholecystectomy	8 cases
Choledochotomy	1 case
Femoral aneurysms	2 cases
Meckel s diverticulum	2 cases
Gastric resection	1 case
Hiatal hernia repair	1 case
Hemangioma liver	1 case

none of the patients in whom major secondary procedures were done in conjunction with aneurysmectomy developed complications related to the additional operations provides good justification for the rationale of this policy In our opinion, therefore, all pathologic lesions discovered in the abdominal cavity which can be treated successfully by operation should be attacked at the time of the laparotomy

MORTALITY

In this series of 101 cases of aneurysm of the abdominal aorta treated by excision, there have been 18 deaths occurring within the first few weeks after operation. Several factors have been largely responsible for this apparently high operative risk. Perhaps the most obvious is the policy of accepting for operation all patients with an abdominal aneurysm almost without exception and irrespective of advanced age, and evidence of cardiac, renal or respiratory insufficiency

The importance of these factors in the mortality is well illustrated by the following observations derived from the analysis of our experience. Thus, the fatality rate in patients in the eighth decade of life was twice that of the patients in the fourth and fifth decades (Fig 6) Moreover, in hypertensive patients the risk was twice that of the normotensive (Fig 7) Review of the causes of death in these 18 fatalities further indicates the significance of cardiovascular and renal disease in these patients in that 14 of the early deaths were due to these causes (Table 2) Moreover, among the 4 patients who recovered from the operation and died several months to over a year later, coronary disease was *the cause of death in 3 of them.*

Still another *important factor contributing significantly* to the mortality is the presence of rupture of the aneurysm. Thus, among the 17 with this complication the operative risk was three times that of the nonperforated group

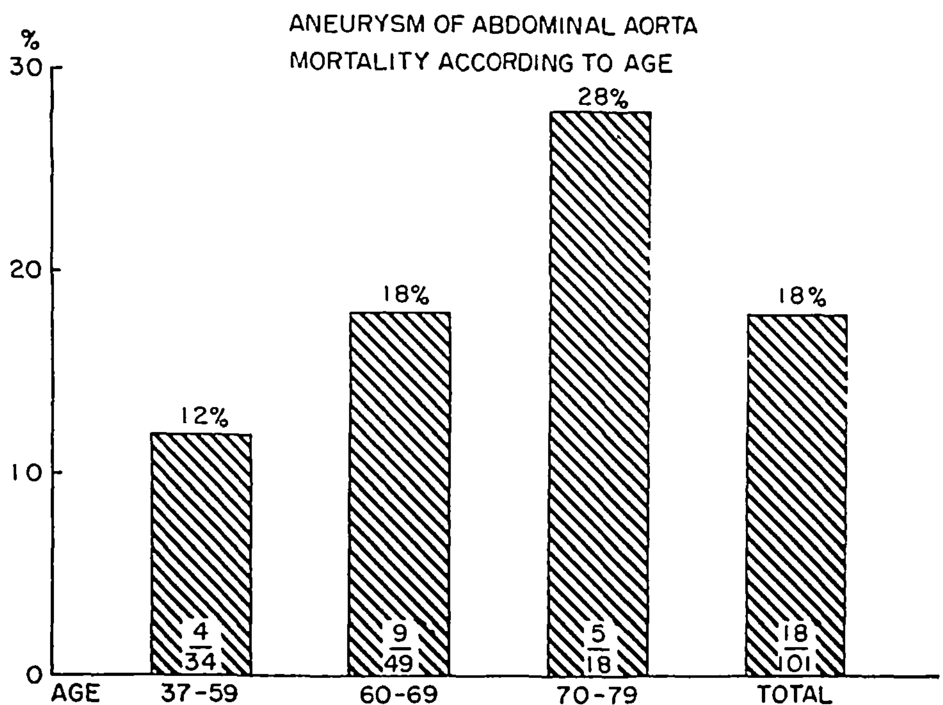


Fig 6. Graph showing mortality rate according to age in 101 patients underg resections of aneurysms of the abdominal aorta

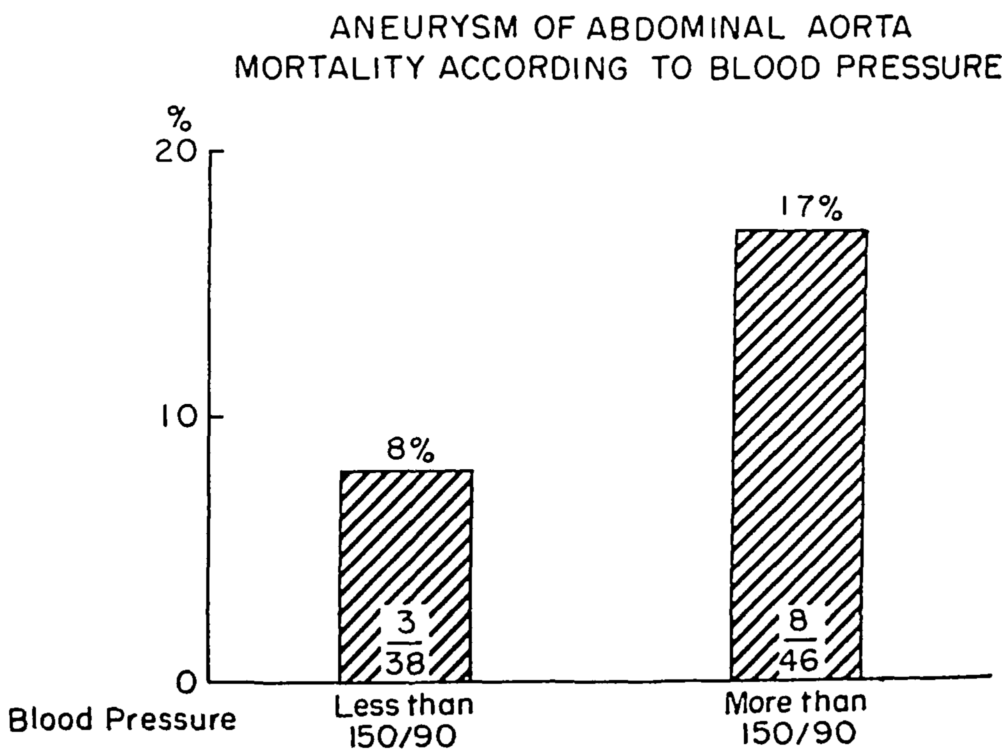


Fig 7 Graph showing mortality according to preoperative level of blood press in 84 patients of the nonruptured group undergoing elective operation for abdomi aneurysm.

(Fig. 8) Most of the operations were emergency procedures performed on patients in shock and with massive retroperitoneal hemorrhage. On the other hand the salvage of 59 per cent of patients in this category is particularly striking In spite of the risk of operation under these circumstances, resection

TABLE 2 ABDOMINAL ANEURYSMS CAUSES OF DEATH (101 CASES)

	Early	*Late*	*Total*
Heart disease	7	3	10
Renal failure	7	0	7
Pulmonary embolism	1	1	2
Rupture aorta above graft	1	0	1
Rupture graft	2	0	2
Total	18	4	22

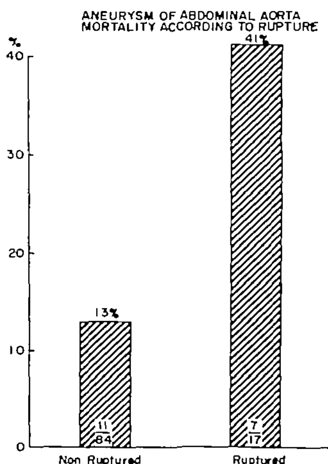


Fig. 8 Graph showing comparison of mortality of patients in the nonruptured and ruptured group in the series of 101 cases of resection of abdominal aneurysm.

of the aneurysm should always be attempted regardless of the apparent hopelessness of the situation. In fact, it is useless to attempt to combat shock in such cases by massive transfusion, and preparations should be made for immediate laparotomy After the proximal occluding clamp is applied, such patients usually respond favorably to blood transfusion and may tolerate the operation itself remarkably well.

With increasing experience in the surgical management of this problem there is reason to believe that the mortality and risk of operation can be reduced significantly. This is reflected by a comparison of the case fatality rates in the first 50 cases of our series with the more recent group of 51 cases. Thus, the operative mortality in the former group was 22 per cent, whereas in the latter it was 14 per cent. The reduction of about 36 per cent of the earlier mortality becomes even more significant in light of further analysis revealing a higher proportion of poor risk cases in the more recent series This is well illustrated by the fact that there were almost twice as many ruptured aneurysms in the more recent series. Among the earlier group, for example, there were 6 cases of ruptured aneurysms with an operative mortality of 66.6 per cent, whereas in the more recent series there were 11 cases with an operative mortality of only 27 per cent. These observations, thus, support the conviction that with increasing experience and improvements in the surgical management of these patients and with the actual technical performance of the operation the mortality will be further reduced.

Nonfatal complications of operation for abdominal aneurysms have in general not been serious In none of the patients has gangrene of the lower extremities occurred, and no amputations have been done Spinal cord damage or visceral damage to organs supplied by vessels originating distal to the proximal occluding clamp has not occurred Follow-up studies for periods extending up to two and one-half years have shown no recurrence of symptoms and maintenance of good function. These observations thus provide mounting evidence for the conviction that this is the most effective form of therapy for this lesion



Question How often is spinal cord damage associated with extensive aortic transplants?

Answer: Our experience with temporary occlusion of the abdominal aorta for insertion of extensive aortic transplants indicates that spinal cord damage is not a significant problem even though the aorta may be occluded for as long as 90 minutes For thoracic aneurysms, particularly those lying near the distal half of the arch, the threat of neurologic sequelae is a real one and in our opinion hypothermia is a useful preventive measure in those cases.

Question How often has it been necessary to sacrifice the inferior vena cava? What complications have been encountered when the inferior vena cava has been ligated?

Answer. We have never sacrificed the inferior vena cava in these cases. We prefer to leave a portion of the aneurysmal sac on the cava if necessary to avoid injuring it Venous bleeding from the cava may be very difficult to manage. Usually we simply oversew the plaque of aneurysmal wall left on the cava and have seen no complications arising from leaving this bit of tissue behind

Question Do you always try to anastomose the hypogastric artery when patent?

Answer If the hypogastric artery is patent distally, I advise that it be reanastomosed if possible.

SURGICAL TREATMENT OF OCCLUSIVE ARTERIAL DISEASE

CHARLES ROB (*London*)

Today I shall confine my remarks to the direct surgical treatment of occlusive arterial disease. The first attempts to restore the flow through a thrombosed artery were made by dos Santos,[1] who introduced the operation of thrombo-endarterectomy, this operation has since been abandoned by many surgeons because of the high failure rate. In my view it is the operation of

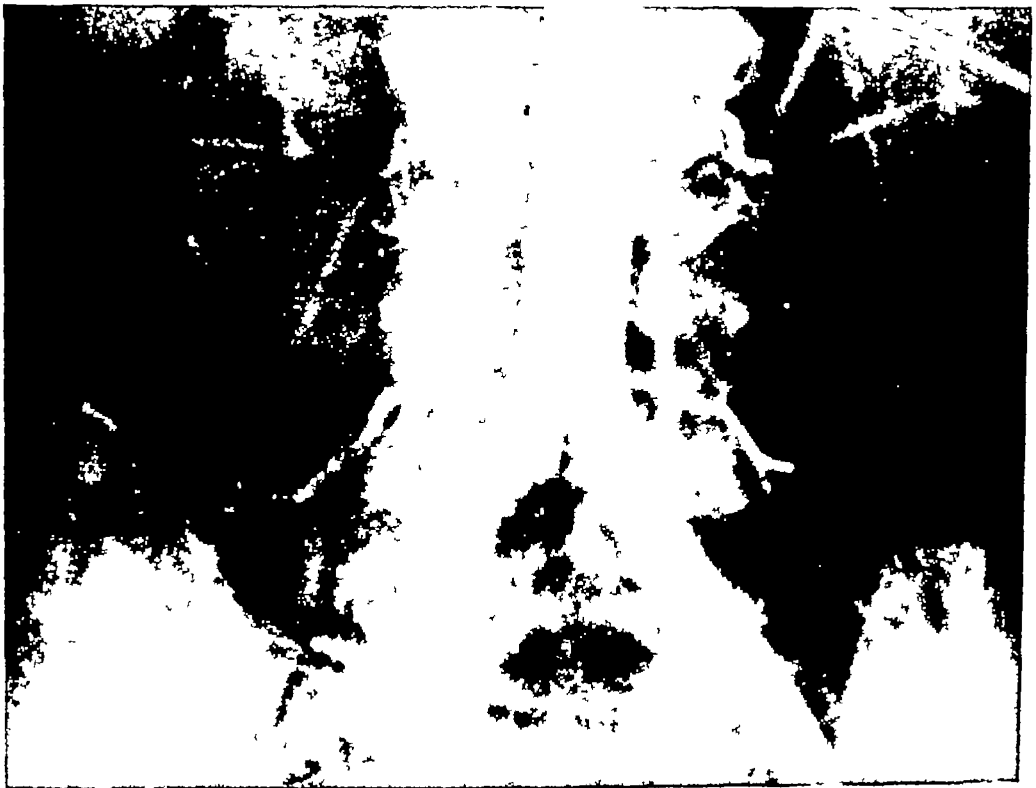


Fig. 1 Aortogram showing an exceptionally well localized occlusion of the right common iliac artery Such a lesion is suitable for the operation of thrombo-endarterectomy.

choice in an occasional patient with an exceptionally short and localized occlusion of a large artery Figure 1 shows such an occlusion in the right common iliac artery of a 46 year old man who was unemployed because his claudication distance was below 50 yards. At operation a pedunculated calcified plaque was removed from the iliac artery and a good result obtained (Fig. 2)



Fig. 2. Postoperative arteriogram from the patient shown in Fig. 1 the localized plaque has been removed.

The key to the success of this operation is careful selection, during the last five years we have performed thrombo-endarterectomy on only 5 occasions, but 4 of these patients are at work with patent vessels

The surgeons of France and Belgium, particularly Fontaine and Hubinot[2] and Kunlin,[3] have had considerable experience of the operations of resection of the thrombosed segment of artery and its replacement by an autogenous vein transplant, or the insertion of a by-pass around the blocked artery with a length of vein anastomosed end to-side to the artery above and below the occlusion. For general use these procedures appear to be superior to thrombo-endarterectomy, but in our hands they have been inferior to replacement of the thrombosed segment by a homologous arterial transplant, and it is upon this that I shall concentrate here.

TABLE 1 93 HOMOLOGOUS ARTERIAL TRANSPLANTS (FROZEN AND FREEZE DRIED)

Left hospital with patent vessels	73 (78.5%)
Thrombosed in hospital	14
Died	6
Operations requiring hypothermia	6

TABLE 2 FATE OF 73 HOMOLOGOUS ARTERIAL TRANSPLANTS PATENT ON DISCHARGE FROM HOSPITAL

Alive and well	52	less than 1 year	22
		1 to 2 years	15
		more than 2 years	15
Subsequent thrombosis in the same artery	13		
Subsequent thrombosis in another artery	6		
Dead	2	1 cardiovascular	
		1 unrelated	

TABLE 3 73 HOMOLOGOUS ARTERIAL TRANSPLANTS PATENT ON DISCHARGE FROM HOSPITAL

49 had obliterative arterial disease
15 further thromboses
1 dead
24 transplants for another reason
4 further thromboses
1 dead (unrelated cause)

During the past five years we have had experience of 93 homologous arterial transplants in patients Table 1 summarizes the early results. It will be seen that 78.5 per cent left hospital with patent vessels. The longer term follow-up is the important thing, and we are now beginning to get some information on the fate of these initially successful transplants. Table 2 shows that a further 19 patients have had another arterial thrombosis, 13 involving the artery in which the transplant had been inserted and 6 another artery, and 2 patients have died, 1 as a result of cardiovascular disease and 1 from an unrelated cause Of the 52 survivors who are alive and well, 24 have been followed for less than 1 year and 30 for longer, the longest a popliteal transplant inserted nearly five years ago. Table 3 gives the proportion of those patients who suffered from obliterative vascular disease, and it will be seen that most of the late complications have occurred in this group Nevertheless, 33 patients with this disease have been relieved of their symptoms and enabled to lead active lives for periods of up to five years from their operations, and a further 15 have obtained temporary relief. The difficulty is to select those patients who will benefit Some idea of the strictness of our selection can be given by stating that of approximately 400 patients with intermittent claudication as their presenting symptom, only 44 have been treated by direct surgery.

Patients with gross generalized arterial disease should be treated in other ways, and any evidence of coronary arterial disease is a contraindication to direct surgery, except to save life or the limb The family history, the patient's age, general health and, in particular, the state of the retinal arteries as observed through an ophthalmoscope, provide valuable help, but the two most important points are the degree of the patient's disability and the length and position of the arterial occlusion. The patient's disability should usually be such that he cannot work. A typical case which should not be grafted is that of the business executive who can do his work but cannot play golf; only in exceptional circumstances would I advise direct surgery for such a

TABLE 4. 64 ARTERIAL TRANSPLANTS IN PATIENTS WITH OCCLUSIVE ARTERIAL DISEASE

Vessel	*Number of Transplants*	*Thrombosed in Hospital*	*Thrombosed Later*	*Patent Today*
Aorta and iliacs	19 (1 died in hospital)	1	0	17
Femorals	26	9	6 (plus 1 dead since discharge)	10
Popliteals	14	1	4	9
Others	5	2	2	1
	3 have thrombosed another artery since operation			

man. As a general rule, the larger the artery and the more localized the disease, the better the result. But fortunately many patients with lesions anatomically suitable for grafting have not sufficient disability to justify the operation. One must never lose sight of the fact that one is treating patients and not arteriograms

THE AORTIC BIFURCATION Thrombosis of the lower abdominal aorta can occur at any age. I have seen it in a child 3 days old and in a woman of

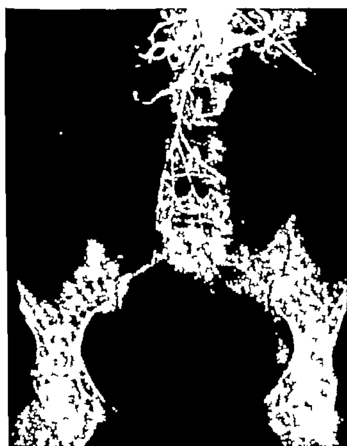


Fig. 3 This patient, a housewife aged 39 was unable to walk for more than a few yards, she was hypertensive and her urine contained albumin, red blood cells and casts. The thrombosis has reached the left renal artery

76, but it is most frequently seen between the 40th and 55th years of life. These patients have a severe disability and many require energetic treatment. We have treated 27 patients with this lesion. The danger to life is that the thrombosis may extend upwards and involve the renal arteries; the risk of this occurring is considerable On the other hand, gangrene is less of a problem and intermittent claudication surprisingly mild in some patients These patients do well with an arterial transplant Figures 3 and 4 illustrate a typical



Fig 4 An arterial transplant has been inserted from the level of the renal arteries to the common iliac on the right and the external iliac on the left side. The operation was performed under hypothermia with a body temperature of 28° C, the aorta above the renal arteries was clamped for 1 hour The patient now does a full day's work and has a normal blood pressure.

case. In no patient have we had a recurrence of symptoms after an initially successful reconstruction of the aortic bifurcation.

THE COMMON AND EXTERNAL ILIAC ARTERIES We have treated 11 patients with occlusions of these vessels with arterial transplants. Ten have done well and are patent today, one has thrombosed Apart from those patients with an associated occlusion of the aortic bifurcation, we have considered bilateral occlusion a contraindication to arterial reconstruction

THE FEMORAL ARTERIES. Occlusive disease is common in these vessels but only a minority will be benefited by a blood vessel transplant The collateral circulation around a short obstruction in the superficial femoral artery is often very good indeed, and occasionally may be sufficient to provide

weak, palpable pulsation at rest in the dorsalis pedis and posterior tibial arteries. We have seen nearly 300 patients with a femoral thrombosis in whom it would have been possible for purely anatomic reasons to have inserted a blood vessel transplant, but we have operated on only 26, 16 left hospital with patent vessels, 6 of these have thrombosed since leaving hospital and 1 has died of cardiovascular disease. An ideal case is illustrated in Fig 5 This patient who was aged 34 had a localized constriction of the



Fig. 5 A stricture of the common femoral artery with a proximal aneurysm in a patient aged 34. The stricture was shown microscopically to be due to occlusive arterial disease (arteriosclerosis) an arterial transplant was inserted.

common femoral artery with an aneurysm proximal, microscopy showed that the constriction was due to arteriosclerosis

THE POPLITEAL ARTERY Thrombosis of this vessel produces severe symptoms which frequently lead to unemployment or amputation. We have reconstructed 14 such vessels, all but one left hospital with patent transplants, but 4 have thrombosed later In our view the rare lesion of primary popliteal thrombosis is a good reason for a blood vessel transplant (Figs 6 and 7). Many popliteal thromboses spread down to involve the bifurcation of this vessel. So far we have only reconstructed this bifurcation on one occasion, this patient has good pulsations in the dorsalis pedis and posterior tibial arteries two years later In some patients with gross arteriosclerosis, reconstruction of the popliteal artery is justified because although the patient has a general disease the thrombosis has caused marked local symptoms Figures 8 and 9 are arteriograms from such a patient.



Fig 6 Fig 7

Fig 6 This patient aged 46 had a thrombosis of the popliteal artery which produced severe intermittent claudication There was little evidence of arteriosclerosis and it was thought to be a primary thrombosis

Fig. 7 The popliteal artery has been reconstructed and the patient can walk for miles



Fig. 8 Fig 9

Fig. 8 Occlusion of the popliteal artery in a patient with marked arteriosclerosis.

Fig. 9 A transplant was inserted into the patient whose arteriogram is shown in Fig. 8, he had crippling symptoms, including rest pain. Three years later he is well with a patent transplant.

OTHER ARTERIES. Five other arteries have been reconstructed with an arterial transplant, comprising 2 brachials, 2 axillaries and 1 subclavian. The brachials and axillaries have thrombosed, 2 in hospital and 2 since discharge. In addition, 1 carotid has been reconstructed by a direct anastomosis; this patient was reported by Eastcott, Pickering and myself in 1954[4] and is of some interest. She was a woman aged 66 who suffered from intermittent attacks of right hemiplegia and blindness in the left eye; arteriography showed a partial occlusion of the left carotid artery (Figs 10 and 11), the attacks were completely relieved by resection of the diseased portion of carotid artery and restoration of continuity by a direct anastomosis.



Fig 10



Fig 11

Fig 10 This partial obstruction to the carotid artery had caused 33 transitory attacks of blindness in the left eye and right hemiplegia Resection of the narrowed segment of artery and reconstruction by a direct anastomosis has prevented further attacks

Fig. 11. The segment of carotid artery resected from the patient shown in Fig 10

SUMMARY AND CONCLUSIONS

In selected patients direct surgery is of great value in the treatment of obliterative vascular disease. Various methods have been employed: in most of our patients a homologous arterial transplant has been used, but there is a small place for the operations of thrombo-endarterectomy and resection with direct anastomosis, in the case of the aortic bifurcation it is probable that prostheses of plastic fabric will replace homologous transplants.

When selecting patients for direct surgery one should exclude, when possible, patients with evidence of coronary arterial disease and evidence of gross generalized arteriosclerosis. The patient's age, disability, family history, general health and also the state of the retinal arteries are important points. The ideal case has a localized occlusion of one artery with relatively normal arteries elsewhere.

In patients with intermittent claudication direct surgery is only required for those who cannot work.

In the case of the aorta and iliac arteries the results have been good. The reason is probably that the operation is technically more satisfactory in these large vessels

The femoral transplants have been the least satisfactory, probably because patients with occluded superficial femoral arteries often have advanced arterial disease.

The popliteal transplants have done well We attribute this to the fact that occlusion of this artery often produces major symptoms in young patients with a relatively normal vascular tree.

Our results with smaller arteries have been less satisfactory, and attempts to reconstruct the dog's coronary artery with an arterial autograft have proved to be a very difficult technical procedure, although some animals have survived.

Probably our most satisfactory results have been in the patient with intermittent attacks of blindness and hemiplegia due to localized occlusive disease of the internal carotid artery, the young patients who were crippled by a popliteal thrombosis, and the patients with high occlusions of the abdominal aorta.

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Question You briefly alluded to coronary artery transplant. Would you discuss it further?

Answer In large greyhounds we have been incising a short section of the main descending ascending coronary artery and inserting a short segment of internal mammary artery in the same animal. A few of our dogs are still alive.

Question Do you have complications of the distal extremities following abdominal aortic grafting?

Answer For the approximately 100 aneurysm cases, no We have had 4 amputations

Question In how many of your cases of dissecting aneurysm was the point of rupture into the outer lumen demonstrated in your dissected specimens?

Answer The answer is three.

ANEURYSMS AND OCCLUSIVE DISEASES OF THE AORTA: ANALYSIS OF 203 CASES TREATED BY RESECTION AND HOMOGRAFT REPLACEMENT

MICHAEL E. DEBAKEY, DENTON A. COOLEY AND OSCAR CREECH, JR. (*Houston*)

Sufficient experience has now accumulated on the use of excision and graft replacement for aneurysms and occlusive lesions of the aorta and peripheral arteries to justify the conviction that it constitutes the most effective form of management for these diseases This is evidenced by the increasing number of reports indicating highly gratifying results following its employment, and is further supported by follow-up observations extending for periods of several years showing maintenance of these good results. Thus, the value and efficacy of this method of therapy may be readily accepted. There remain, however, a number of factors, both technical and physiologic, that have an important bearing upon the feasibility of the procedure and, indeed, even restrict its successful application under certain circumstances. Accordingly, a consideration of some of these factors based upon an analysis of our experience seems desirable.

During the four-year period since our first successful resection of an aneurysm of the aorta, we have employed the procedure of excision with aortorrhaphy or with graft replacement in more than 250 cases of aneurysms or occlusive lesions of the aorta and peripheral arteries. Of this number 218 involved the aorta, 49 of which were located in the thoracic and 169 in the abdominal regions. The most common lesion in this series was aneurysm, being present in 35 of the former and 101 of the latter. All of the others were occlusive lesions (Table 1). Analysis of our experience with aneurysms of the abdominal aorta is reported elsewhere in this symposium and will not be considered here. Similarly our experience with the preparation and with the fate of homografts and other arterial substitutes is presented in another report in this symposium.

Among the 49 cases of thoracic aortic lesions 15 were sacciform, 14 were fusiform and 6 were dissecting aneurysms. The remaining 14 cases were occlusive lesions, being congenital coarctations in 12, 2 of which were associ-

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ated with aneurysm formation, and acquired in 2 cases Because of the length of the constricted segment or the association of aneurysms, homograft replacement following resection was necessitated in all of these latter cases Owing to the presence of a well developed collateral blood supply the problem here, in contrast to aneurysms, is relatively simple and unassociated with the ischemic hazard of temporary arrest of the circulation. The problem is primarily technical, consisting essentially in the excision and replacement of the occlusive segment by proper homograft. Emphasis should be placed on complete restoration of all major vascular channels, including particularly the left subclavian artery which is not infrequently involved in the obliterative process All of these patients obtained excellent results

TABLE 1 RESULTS FOLLOWING RESECTION AND HOMOGRAFT REPLACEMENT IN AORTIC DISEASE

Type of Aortic Disease		*No Cases*	*Recovered*		*Died*		
			Excellent	Poor	Early	Late	Total
Aneurysms							
Thoracic	Fusiform	14	9	0	5 (35%)	0	5
	Dissecting	6	4	0	2 (33%)	0	2
Abdominal	Nonruptured	84	72	0	11 (13%)	4	14
	Ruptured	17	10	0	7 (41%)	0	7
Occlusive Disease							
Thoracic		14	14	0	0	0	0
Abdominal		68	59	4	2 (3%)	3	5
Total		203	168	4	27 (13%)	6	33

In fusiform aneurysms of the thoracic aorta, however, the problem is much more difficult and hazardous, owing primarily to the ischemic effect on the tissues during the period of temporary interruption of aortic circulation. This is influenced by a number of factors, but the most important are the level and duration of occlusion and the length of the segment to be excised. The highest level of occlusion in our experience was just distal to the origin of the left common carotid artery, and at this level the most vulnerable tissue to ischemic damage is the spinal cord. Indeed, according to our experience there was no evidence that temporary occlusion of the aorta for periods up to about 1 hour produced ischemic damage to any other organs Thus prevention of spinal cord damage constitutes the major problem in the resection of aneurysms arising at levels up to the left common carotid artery

To overcome this problem several approaches may be used. These include the use of temporary shunts around the occluded segment of aorta, hypothermia, and certain steps in the operation designed to minimize the period of circulatory arrest. In our experience major emphasis has been placed upon the last two methods, since the shunting procedure appeared to add to the complexity of the operation. Indeed, the impression has been gained that simplicity and expeditious performance of the procedure constitute impor-

tant practical factors in the success of the operation. Moreover, for located on the descending thoracic aorta no higher than the sixth or s thoracic vertebra these technical considerations seem adequate. Thus, the 4 cases in our series with lesions located at such levels, the procedu successfully performed in 3. The one death in this group occurred fro den hemorrhage on the eighth postoperative day resulting from a tear graft beginning at the line of anastomosis. Although neither shunts nor thermia were used during the operative procedure in these patients, showed any evidence of neurologic disturbances or ischemic damage to organs following temporary arrest of aortic circulation for periods v from 24 to 60 minutes.

Among the remaining 10 cases with lesions located at higher level requiring aortic occlusion in the distal part of the arch, hypotherm employed in 6 Although 2 of these patients died, one from septicem the other from secondary hemorrhage, none manifested any evidence chemic damage to the spinal cord following aortic occlusion at such levels for periods up to 62 minutes. In the other 4 cases in this group thermia was not used, and it would appear significant that manifestati spinal cord damage occurred in 3 of the patients. Fortunately, these ch were mild and transient in 2 but probably contributed to the death third patient. The fourth patient in this group died within a few hours operation, presumably of heart failure. Thus, these clinical observa which conform with our experimental findings, strongly suggest that thermia provides protection against spinal cord damage following temp aortic occlusion up to one hour. Accordingly, its usefulness for this pu is particularly indicated in the excision of the more proximally situated rysms of the descending thoracic aorta or of the distal part of the arch

Dissecting aneurysms of the aorta constitute an entirely different pr from that of sacciform or fusiform aneurysms, both on the basis o pathologic characteristics and the surgical approach. The dissection comn begins a few centimeters above the aortic valves or in the descending th aorta near the origin of the left subclavian artery, probably through in the intima. Once this occurs, separation of the intramural layers o aorta by the forceful stream of blood produces dissection usually a junction of the middle and outer thirds of the media, and progresses di involving all or a portion of the circumference of the aorta. As branche encountered, they may be sheared off or the dissecting process may e along them for varying distances, thus diminishing or completely interru the blood supply to these areas. The extent and course of the dissecti quite variable. In the most acute and severe form there is rapid dissection finally terminal perforation through the adventitia into the pericard mediastinum, pleural or peritoneal cavities with death in a few hours or In the subacute type the process takes place gradually over a period of or weeks with terminal adventitial rupture and death. In the chronic reentry of the dissected passage into the lumen of the aorta takes place, forming a "double-barreled" aorta. Under these circumstances the passage may become covered with endothelium or occasionally may be

literated by thrombus formation with subsequent fibrous tissue organization. The extent of the dissection may be relatively limited or it may involve the entire aorta and even extend down to the popliteal and tibial arteries The gravity of this condition is demonstrated by the fact that less than one fourth of the patients survive the initial attack.

Treatment of this condition has been largely symptomatic and unsatisfactory The problem is concerned essentially with the prevention of further intramural dissection and terminal rupture through the outer adventitial layer and restoration of blood flow through the normal lumen. On the basis of our

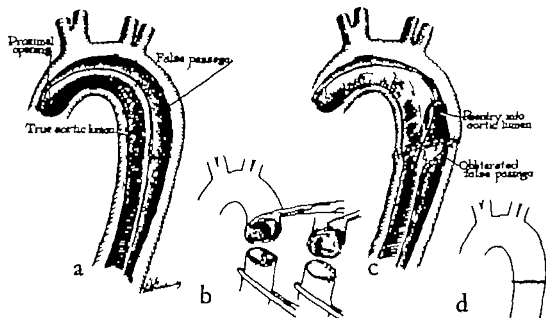


Fig. 1 Drawing showing technique of surgical procedure used in dissecting aneurysm of aorta. *a*, Sagittal view of thoracic aorta showing proximal opening in ascending arch and "double-barreled" lumen produced by dissecting aneurysm. *b*, After cross-clamping and dividing descending thoracic aorta the false passage distally is obliterated by approximating the outer and inner layers, and a small window is created in the proximal internal layer by excision of a wedge-shaped segment to permit reentry from the outer lumen. *c*, Sagittal view showing completed operation following end-to-end anastomosis of the divided aorta and illustrating principle of diverting blood from double lumen with common opening proximally into single normal lumen distally *d*, Diagrammatic drawing showing external appearance of completed operation following end to-end anastomosis of aorta.

experience with 6 cases it is believed that this may be achieved by one of several surgical approaches In cases in which the dissection begins in the ascending or descending part of the aortic arch, the most logical procedure would seem to be the creation of a reentry passage into the aortic lumen above with obliteration of the false passage below This may be accomplished by cross-clamping the descending thoracic aorta, dividing it completely between the clamps, obliterating the false passage below by approximating the outer and inner layers, excising a small segment from the inner intimal layer above to produce reentry of the outer lumen, and then completing the procedure by end to-end anastomosis (Fig 1) Thus, blood flow from the double aortic

lumen above is diverted into the single normal lumen below. Subsequent thrombus formation and fibrous organization may then take place in the distal false passage to obliterate it. In some instances it may be desirable to excise a segment of the involved aorta owing to the occurrence of fusiform aneurysmal dilatation in the outer wall and then bridging the defect if necessary by a homograft. Under these circumstances the principle of obliterating the distal false passage should be applied. This latter procedure was employed in 2 of our 6 cases. One was successful, but the other patient died from ventricular fibrillation occurring during hypothermia. In 3 of the cases the former procedure was employed with one death. This occurred suddenly on the eighth postoperative day from rupture of the aneurysm into the pericardial cavity. The last case was rather unusual in that the dissection was small and well localized and it was therefore possible to excise it completely with repair by lateral aortorrhaphy, thus providing still another but probably uncommon approach to the problem. The 4 patients that recovered have remained well, and although the follow-up periods are relatively short, the longest being slightly less than one year, these early results are considered encouraging

Additional experience with occlusive or thrombo-obliterative disease of the abdominal aorta, now totaling 68 cases, provides further support of a number of observations previously reported as well as certain modifications in the technical procedure designed to restore peripheral circulation more effectively. Thus, age and sex incidences, as well as pathologic features and clinical manifestations, did not differ significantly from those which we previously reported. All but one of the patients were males, and only 3 were Negroes. The youngest was 33 and the oldest was 66 years of age Most of the patients (71 per cent) were in the fifth and sixth decades, although an appreciable number (22 per cent) were in the seventh decade Of possible significance is the fact that the patients with complete occlusion of the aorta were on the average somewhat younger than those with incomplete occlusion

The characteristic clinical manifestations of arterial insufficiency of the lower extremities were present in all the cases, but the nature and extent of the occlusive process is best demonstrated by translumbar aortography From these studies and the findings at operation, two patterns of the disease are suggested, namely, partial and complete aortic occlusion. The former type was observed in 30 and the latter in 38 cases Differentiation between these two forms of this disease is further suggested by other findings in the analysis. Thus, clinically hip, thigh, and buttock pain as well as sexual impotence was much more commonly encountered among the patients with complete than among those with incomplete occlusion Similarly there was a somewhat greater tendency for patients with complete aortic occlusion to have an associated hypertension In 44 per cent of the entire series there was an elevated blood pressure, i e., greater than 150 mm Hg systolic or 90 mm Hg diastolic Perhaps the most significant difference between these two types of the disease lies in the extent of the occlusive process, particularly in the peripheral arterial bed. The impression has been gained that in cases with incomplete aortic occlusion there is a more frequent occurrence of peripheral arteriosclerosis obliterans. By the same token, in cases with complete aortic occlusion there

is a greater tendency for the occlusive process to be better localized to the terminal aorta, bifurcation, and iliac vessels This is further supported by the better results obtained following operative treatment, suggesting some prognostic significance to this factor

Additional experience has supported our earlier conviction that excision of the involved segment of terminal aorta and bifurcation with replacement by homograft is the procedure of choice in the great majority of cases Indeed only 4 cases in our series have been treated by thrombo-endarterectomy alone and all of these were incomplete aortic occlusion. Thrombo-endarterectomy is, however, a useful and effective procedure to apply to the proximal aortic and distal iliac segments after excision of the primary occlusive process about the bifurcation. This is particularly useful in the proximal aortic segment in order to remove the partially organized thrombotic process which may extend



Fig. 2. *a*, Preoperative aortogram in case of Leriche syndrome with incomplete occlusive disease and complete occlusion of left common iliac artery *b* Postoperative aortogram showing restoration of normal blood flow following resection of terminal aorta and bifurcation and replacement with bifurcation homograft and use of by-pass procedure on left as illustrated in diagram *c*.

up to and even slightly above the renal arteries, as well as to conserve a sufficient margin of aortic wall below the renal arteries to permit anastomosis to the graft. In cases in which the occlusive process is extensive distally and involves virtually the entire iliac vessels, thrombo-endarterectomy has been found less effective and associated with a high incidence of subsequent thrombosis Indeed not infrequently in such cases the occlusive process is so firmly fixed that no line of cleavage can be obtained. Under these circumstances we have more recently employed a by-pass procedure with the insertion of another segment of graft between the bifurcation homograft and the femoral artery below (Fig 2) An end-to-end anastomosis is used to attach this segment of graft to the iliac portion of the homograft above, and an end-to-side anastomosis is done to attach it to the femoral artery below Although this procedure is not often necessary, it has been found particularly useful under the circumstances described and has proved successful in every case in which it was employed Although bilateral lumbar sympathectomy was routinely em-

ployed in our earlier experience, more recently this additional procedure has been done only occasionally and in cases in which there is some associated peripheral arteriosclerosis obliterans.

The risk of operation in this group of patients is much less than that associated with aneurysms of the aorta, owing probably to the fact that the patients are somewhat younger and in general have less severe associated cardiac and renal disease. There were, for example, only 2 operative deaths, one from cardiac and renal failure and the other from a post-transfusion bleeding tendency (Table 1). Three of the patients died later, one from heart failure, one from septicemia and one from secondary hemorrhage due presumably to rupture of the iliac artery just distal to the line of anastomosis to the graft

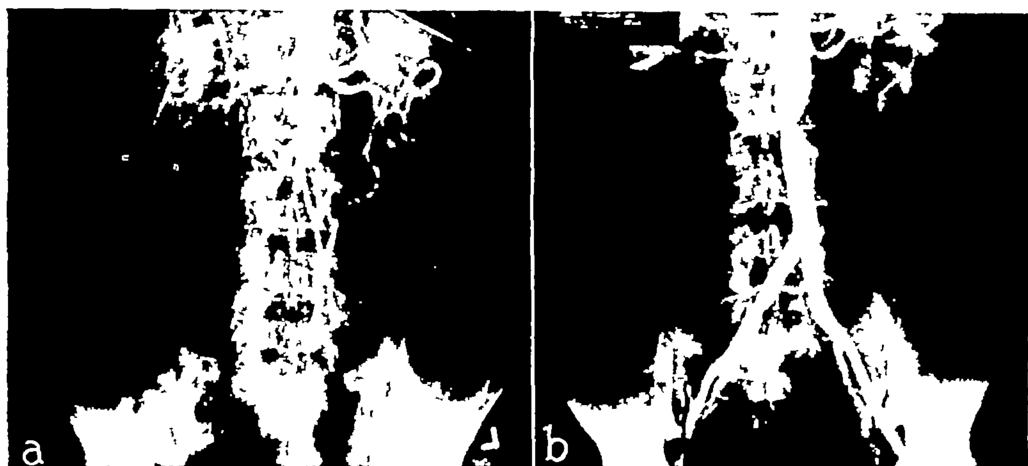


Fig 3 *a*, Preoperative aortogram in case of Leriche syndrome showing complete occlusion of terminal aorta *b*, Postoperative aortogram made almost two years after resection and replacement with bifurcation homograft showing restoration of normal blood flow The patient has remained well with good pedal pulses.

All but 4 of the remaining patients have obtained definite improvement in their condition In these 4 cases amputation of one extremity was subsequently necessary, and it appears significant that in all of them extensive thromboendarterectomy was required and that they all had an appreciable degree of peripheral arteriosclerosis obliterans. Moreover, 3 of these 4 cases had incomplete aortic occlusion, lending support to our previous impression that this type of occlusive process is more commonly associated with peripheral arteriosclerotic disease. This impression is further supported by the fact that in general better results were obtained in the group with complete aortic occlusion. Thus of the 38 cases in this group all but 2 had complete restoration of pulses, an incidence of 95 per cent, whereas among the 30 cases with incomplete aortic occlusion, this incidence was 80 per cent. We are inclined to believe now that under the circumstances encountered in the 4 cases that subsequently required amputation as a result of postoperative thrombosis of the endarterectomized iliofemoral segment, the by-pass procedure should be employed Significantly, this complication has not been encountered during our more recent experience with the last 30 cases.

Follow-up studies on these patients extending up to approximately two years have been most gratifying and have revealed maintenance of the good results obtained immediately after operation. In some of the patients aortography has been repeated a year or more after operation and has revealed no significant changes in the patency of the graft (Fig. 3) Patients who were forced to stop work have resumed their normal activities, including in some cases hard labor Significantly, too, many of the patients who complained of sexual impotency have expressed gratification for the return of sexual function.

RAPID VESSEL ANASTOMOSES WITH TRANSIENT ARTERIAL INTERRUPTION EMPLOYING SUTURE METHODS OVER A REMOVABLE BIVALVE PROSTHESIS

James D. Fryfogle, John T. Small, Walter Stenborg and Carl Samberg (*Detroit*)

The relief of obstruction is a common duty of a surgeon and this fact is evident in many cardiovascular problems. Sheer speed was important in pre-anesthetic days, and even now there are times when rapidity of action is essential. Mammalian tissues and organs exhibit a varied tolerance to complete interruption of their blood supply, a condition which may be encountered in the repair or replacement of major arterial trunks. Successful anastomoses require careful, time-consuming techniques. We present in this preliminary report a method of making end-to-end anastomoses with minimal interruption of the circulation, using a removable cuff type of prosthesis and making it possible for the suturing to be carried out in a leisurely manner.

The work of Gross and his group[1] with preserved homologous arterial grafts in 1947 reopened a field of blood vessel surgery which had been begun by Carrel[2-5] in the early part of the century The first arterial grafts placed in the human aorta were used by Gross to bridge the defects encountered when the long narrowed segments of certain coarctations were excised. Deleterious effects were not produced by clamping the thoracic aorta because of the collateral circulation which is invariably present in this condition. The presence of an aneurysm in the thoracic aorta may increase the safe period of cross-clamping, because DeBakey and Cooley[6] did not observe neurologic signs when the lower thoracic aorta was occluded for 45 minutes. On the other hand, the patient of Lam and Aram had[7] some sequelae of spinal cord ischemia when the aorta below the subclavian artery was clamped for only 24 minutes. Evidently, the abdominal aorta can be clamped for very long periods without damage to the tissues supplied by it. The record in this respect seems to have been established during an operation by Moore[8] when the abdominal aorta was occluded for 5 hours and 30 minutes.

Obviously, the period of arterial occlusion during an anastomosis could be reduced by the utilization of some nonsuture technique, such as with the Blakemore vitallium cuffs[9-10] which gave promising results when used with

vein grafts to reestablish the continuity in arteries of the caliber of the femoral. The occlusion time can also be reduced by temporary intubation, the tube being removed just before the completion of the suture line. This method was suggested by Carrel,[11] revived by Hufnagel[12] and first used clinically by Lam and Aram.[7] Shunts attached laterally to the aorta and/or its branches can be arranged. Hardin and Schafer[13] used multiple shunts of polythene tubing in their attempt to resect and replace the arch of the aorta. Izant, Hubay and Holden[14] tapped the distal and proximal aorta with metal prostheses made like the valve stems of automobile inner tubes The "stems" were connected by plastic tubing during the by-pass.

In the summer of 1951 in Mt. Carmel Mercy Hospital Experimental Laboratory, Dr Walter Stenborg, in attempting to bridge arterial defects with

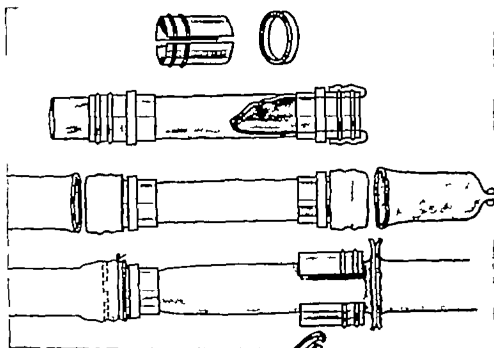


Fig. 1 Showing steps in arterial anastomosis over the removable prosthesis.

venous grafts, devised the original couple. Its use with vein grafts was considered unsatisfactory because of the technical difficulty in suture. When arterial homografts became available, the couple was redesigned by one of us* so that the edges were raised rather than grooved, thus permitting a palpable line for suture placement. The plastic ring was substituted for hinges and an instrument was made for easy placement of the couple (Fig. 1)

As one examines the total aorta at autopsy it is evident that all of its major branches, the innominate, carotid, subclavian, celiac, superior mesenteric, renal, iliacs and the aorta proper, lend themselves to the application of couples that range in size from 2 to 11 mm. in diameter The dissection of these arteries presents problems in the available length that is needed to form an adequate cuff. Thus, although the lumens are adequate it is necessary to have couples

*J D F

of different lengths. These vary from 1.75 to 2.2 cm. For example, should the innominate's base be short or bifid, a small couple may be used on the carotid and the subclavian clamped and temporarily by-passed. The celiac is almost always short as are the renals, particularly the right. Care in removing the graft and insuring adequate length of these vessels is essential.

The couples are applied by the placement of traction sutures to the cut edge of the graft, threading them through the couple and pulling the vessel through. The same traction sutures can be used to cuff the vessel on the couple. A fine silk ligature secures the cuff between the plastic ring and the second ridge of the couple.

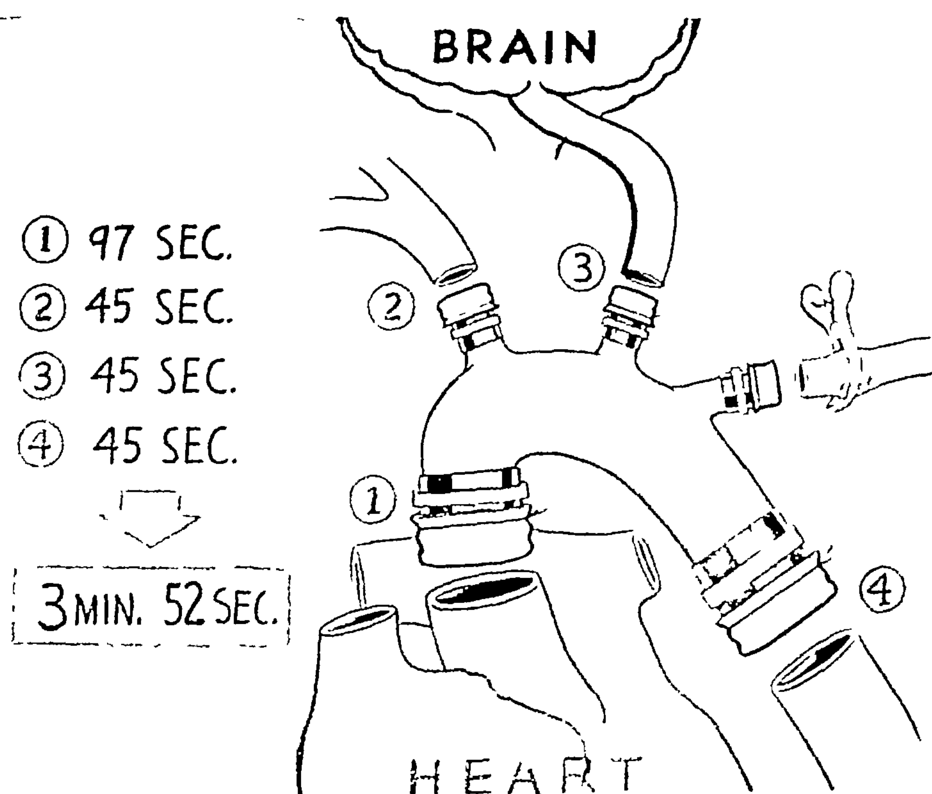


Fig 2 Use of couples in replacement of aortic arch

As many couples are applied to the graft as there are limbs that need immediate blood supply. In the replacement of the entire arch we have found it practical merely to occlude the left subclavian and sometimes the right if the innominate is bifid or has a short stump and to clamp across the aorta between the carotids. Thus, after insertion of the aortic and right carotid couples, the brain receives blood in a period under 3 minutes. An individual couple requires approximately 45 seconds to insert and the time from the occluding clamps, plus the longitudinal slitting of the vessels and the placement of a single stay suture in the upper edge of the host vessel, is included

The method of insertion, particularly in the presence of aneurysm or of coarctation, is of extreme importance. With the graft prepared and the couples in place the diseased portion of the vessel is dissected free, occluding clamps are placed above and below the pathologic segment, and a longitudinal inci-

sion of the segment and all of its branches is made, being careful to leave a length of vessel adequate to cover the cuff at each limb to be grafted. At this point the incision is made horizontal to the occluding clamp approximately two-thirds around the circumference of the diseased vessel, leaving one half of the vessel wall intact on the posterior surface. This maneuver accomplishes three things. (1) it lays wide the area of your dissection permitting free movement of the graft and its immediate positioning; (2) it holds the cut ends of the host vessels so that they do not retract; (3) in sewing, the cut edge of the diseased vessel can be used for traction so that complete rotation is possible for suturing on the posterior wall.

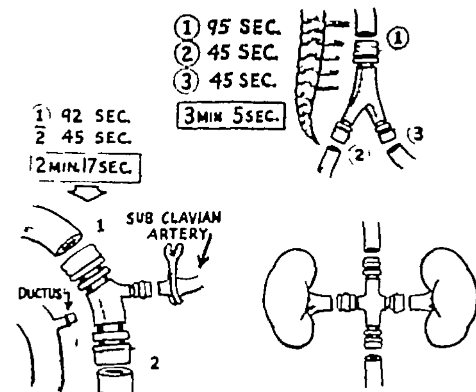


Fig. 3 Use of couples in aortic replacement in several locations.

With the release of the occluding clamps and the reinstitution of oxygenated blood to important viscera, the suturing may be done at leisure. We have found that interrupted sutures do not give a satisfactory result. The area of the suture line is well demarcated and can be felt with the finger The raised edges are 1/8 inch apart allowing ample room for a running, back-tracking suture from one side of the groove to the other

Upon completion of the suturing, the tie holding the two intimas can be cut, the plastic ring cut with a heavy scissors and the entire couple removed. The edges can be trimmed and the shell of the diseased vessel cut away

Despite the fact that the suture of the under surface is done blindly, i.e., on the graft side, we have been well pleased with the suture lines examined in animals after different time intervals (Fig. 5)

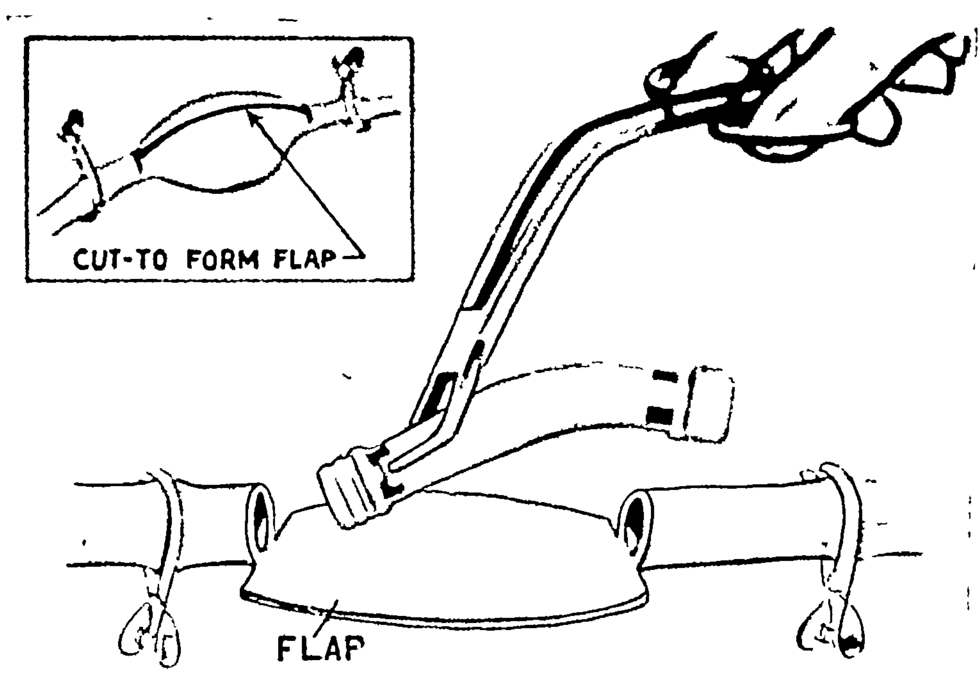


Fig 4 Method of placement of graft to replace aneurysm.



Fig 5 Appearance of suture line two and one-half months after the insertion of aortic graft in a dog

SUMMARY

1 A simple removable vascular splint applicable to a variety of lesions in the vascular tree is presented.

2. Avoidance of hypothermia, complex shunts, extracorporeal pumps and haste in the formation of suture anastomoses is made possible.

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DISCUSSION

Chairman DeBakey

As most of you know, Dr Dubost deserves considerable credit for having done the original case of aneurysm of the abdominal aorta by resection and graft replacement. It is good to know that the patient on whom he did this first operation, approximately four years ago, is doing well. It is heartening.

Charles Dubost (*Paris*)

If the possibilities of resection of the aneurysms of the thoracic aorta are still restricted, it is quite a different matter at the level of the abdominal aorta. Here, the resection of the aneurysms, followed by the reestablishment of continuity by grafting, constitutes at the moment the choice therapeutic method which has proved itself since the first case which we operated on in March 1951 This patient is still living

The intervention remains, however, a major operation, the risks of which are important. It should not even be considered except for those cases in which the patient is in a good cardiovascular state and is not too advanced in age. For those aged above 70 the intervention offers a greater risk with a higher percentage of deaths than it does for those patients who are younger. The volume of the aneurysm will also have to be taken into account as well as the upper limit of its size as defined by the aortogram. Exploratory intervention alone can determine the connection of the tumor with the neighboring organs, define the upper limit of the ectasia and its relations with the origin of the renal arteries, and evaluate the extension of the atheromatous process on the iliac arteries. If the subrenal aorta is infiltrated with calcareous matter, or if it is of a doubtful quality, then the intervention should be abandoned because of the immediate difficulties of doing the anastomosis, and the risks of an eventual rupture. In the same way, important lesions of the iliac arteries can give rise to the fear of a secondary thrombosis of the graft in the future. There again, a resection at any price should take second place to any other palliative method.

OPERATIVE METHODS. First of all the approach should be wide and give sufficient exposure of the upper aorta and the iliacs. For these reasons we prefer to use the transperitoneal median route. In certain cases the duodenum can be adherent; in one of our cases it happened to be intimately merged with the aneurysmal sac, and we had great difficulty in liberating it without rupturing the aneurysm. The lower vena cava and the left common iliac vein may also prove difficult to separate In certain cases it may even be advisable to leave a fragment of the aneurysm wall in contact with the vein, rather than to risk a wound of its wall, as it is always difficult to effect its repair.

The reestablishment of the continuity is obtained by a graft of human aorta, preserved according to Gross or deep-frozen. We have had no experience with the plastic prostheses used by Blakemore and by Shumacker. We think, however, that their application would, if the results should prove to be satisfactory, constitute an extreme simplification of this important problem.

In our last 4 cases we were confronted with a subrenal aorta of doubtful

TABLE 1. ATHEROSCLEROTIC ANEURYSMS OF THE ABDOMINAL AORTA

Age	*Sex*	*Operation date*	*Approach*	*Clamping min*	*Graft*	*Results*
51	M	3/51	Extraperitoneal abdominothoracic	60	Gross	Excellent 4 years
50	M	12/52	Left extraperitoneal	50	Gross	Excellent 2 years
59	M	7/53	Left extraperitoneal	50	Gross	Excellent 18 months
55	M	10/54	Midline laparotomy	40	Deep freezing	Death 12th day
60	M	11/54	Midline laparotomy	70	Deep freezing	Excellent
65	M	12/54	Midline laparotomy	120	Deep freezing	Excellent

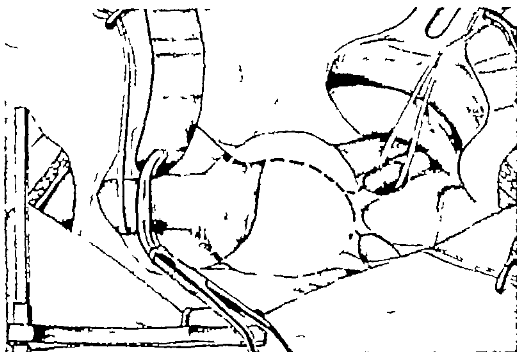


Fig 1 Dissection of aneurysm of abdominal aorta.

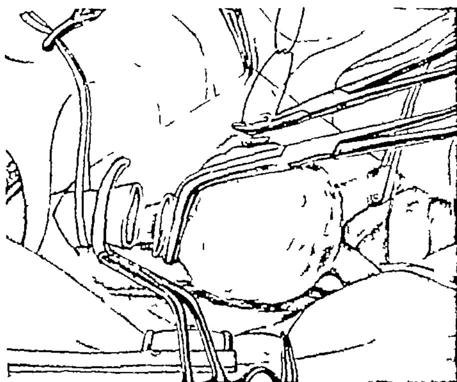


Fig. 2. The upper part of the aorta is cut just above the aneurysm permitting dissection from above to below

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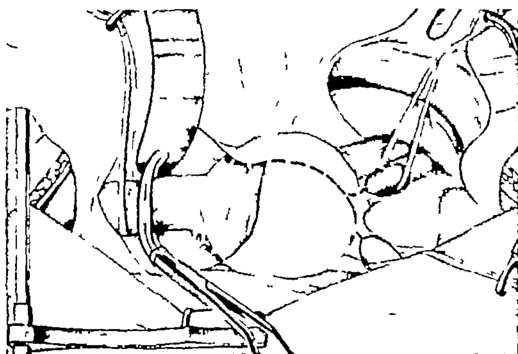


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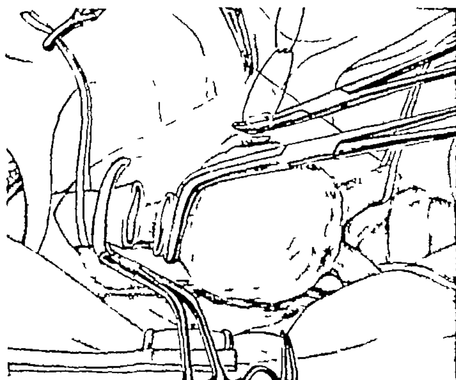


Fig. 2. The upper part of the aorta is cut just above the aneurysm permitting dissection from above to below

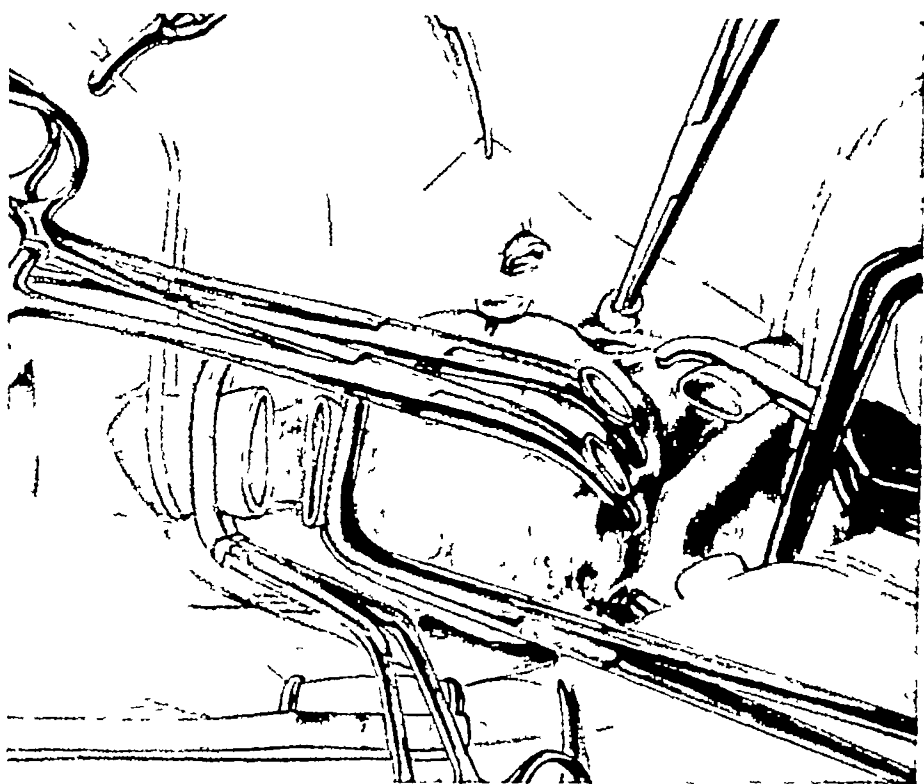


Fig. 3. After cutting the iliac arteries when the dissection from above to below is difficult, one can begin dissection from below to above.

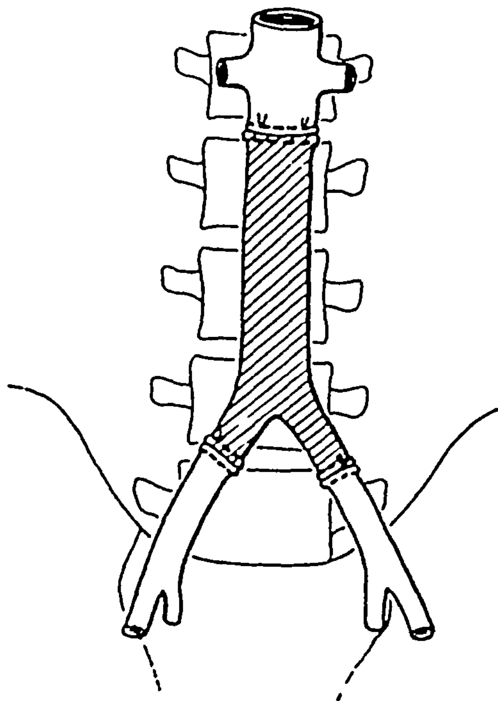


Fig 4

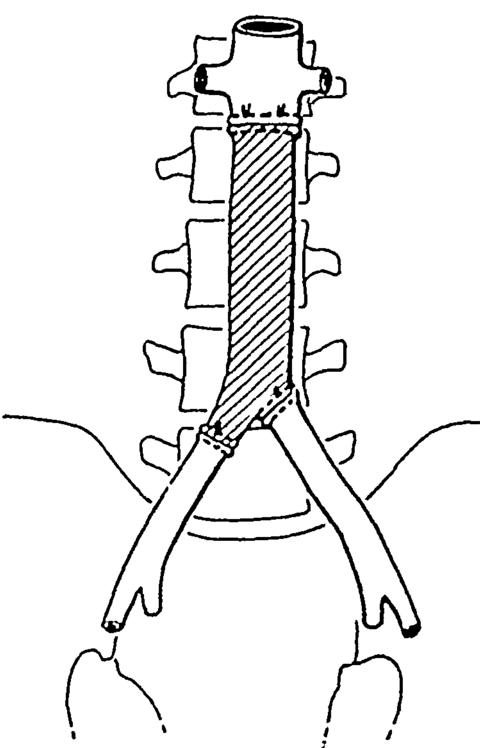


Fig. 5

Fig 4 Y-shaped graft of abdominal aorta.

Fig. 5 Straight graft with end to end anastomosis between end of graft and right iliac artery, and end to side anastomosis with left iliac artery.

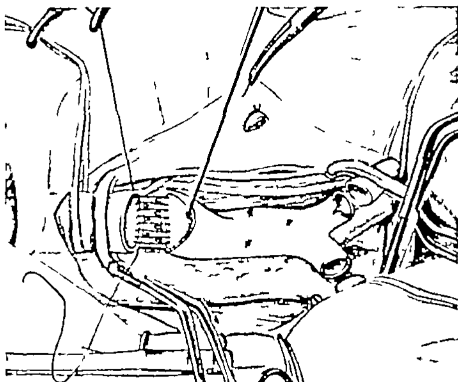


Fig. 6. The graft in place, showing placing of upper suture line.

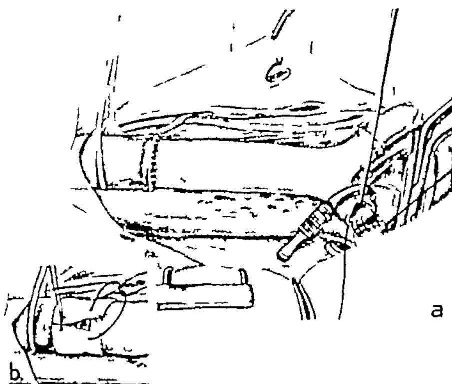


Fig. 7 *a* At the level of the two anastomoses between the end of the graft and the iliac artery *b* It is a good precaution to put around the upper anastomosis a center graft, especially when the wall is in poor condition. This may be a means of averting a second rupture at this level.

quality and we thought of wrapping the upper anastomotic zone in a protective sliding cylinder of graft which could constitute a successful protection against secondary leakage in the suture zone. This external tube of graft tissue tightly encircled the area of the anastomosis and was fixed by a few stitches to the aortic adventitia and to the graft.

RESULTS. Using this method we have operated on 6 cases of aneurysm of the subrenal abdominal aorta, 5 of which were successful. The operations took place 4 years ago, 2 years and 3 months ago, 1 year and 8 months ago, and two of them 4 months ago.

We lost one patient on the fifteenth postoperative day, through a severe pulmonary embolism after a futile Trendelenburg operation.

Josephus Luke (*Montreal*)

In his very fine presentation Mr. Rob mentioned the fact that he rarely uses the operation of thrombo-endarterectomy. I would like in this short discussion to mention our experiences with this procedure, and to tell you why I feel that it is still a good one. I feel it has been thrown aside by the majority of vascular surgeons much too soon.

Following the lead of Raboul and later Wylie, we began this operation nearly three years ago. Before the institution of our vessel bank we had done 6 cases. Four of these were in the aorta and/or common iliacs. Of these 6 cases, 4 were completely successful with return of pulses. In one the pulses returned in one leg and not in the other, although the poor leg was improved by a cross circulation of the internal iliacs. One was a failure owing to infolding of the distal intima.

Encouraged by these successes, and attempting to forget our failure, we have continued this procedure but have drawn up some indications and contraindications for it. At the present time we believe that thrombo-endarterectomy is an excellent alternate to grafting in major vessels, and in my opinion it is a preferable operation. The contraindications for thrombo-endarterectomy are major calcification in vessel wall, marked thickening of the intima, and extensive degenerative changes throughout the major vessel.

At the present time when an area of segmental occlusion is approached, we routinely begin a thrombo-endarterectomy. We have the area isolated between the clamps. It is a simple thing to make a longitudinal incision, assess directly what the vessel wall is like, the degree of atheromatous change, and the thickening of the intima.

If any of our contraindications are met, then the case is approached with a graft on hand, and grafting is carried out. We find following these indications and contraindications that we will do about 50 per cent of the cases by thrombo-endarterectomy and the other 50 per cent by grafting.

In the past year and a half we have had only one more failure of thrombo-endarterectomy to completely restore the peripheral pulses. There is one difficulty that I would like to mention in thrombo-endarterectomy, and that is the infolding of the distal cut intima. The one failure that resulted in bilateral thigh amputation we feel was due to this cause. In fact, we know

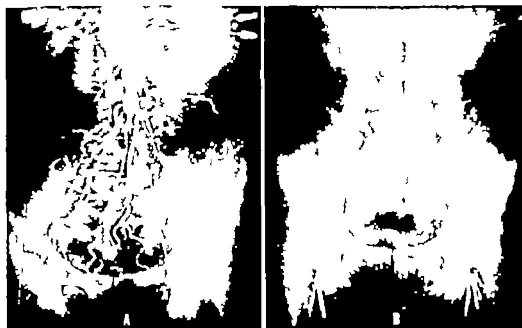


Fig. 1 *A*, Preoperative aortogram revealing extensive aortic and bilateral common iliac segmental occlusion in a 41 year old woman. *B*, Postoperative picture following thrombo-endarterectomy revealing failure to open the right common iliac because of technical difficulties. However, the claudication distance in the right leg has improved from 100 feet to 500 yards.

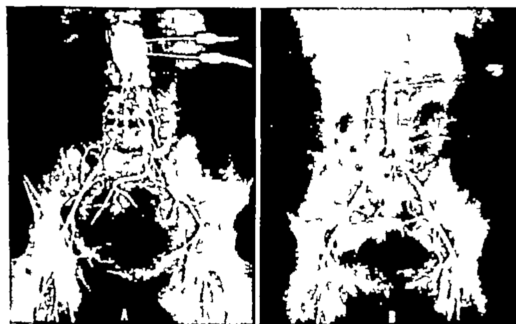


Fig. 2. *A* Preoperative aortogram showing thrombotic occlusion of the lower end of the aorta and both common iliacs. *B*, Postoperative aortogram following operation of lower aortic and bilateral common iliac thrombo-endarterectomy Complete restoration of peripheral flow resulted. Follow up two years.

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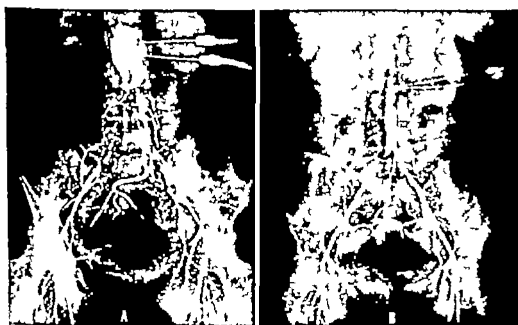


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it was, because we reoperated on the patient three hours later and found the folded intima occluding the vessel.

In thrombo-endarterectomy, our experimental work has shown that the vessel is re-endothelialized in about ten days, and therefore, in essence, after ten days you have what amounts to an autograft—certainly a preferable thing to the unknown fate of the present homografts.

The fixation problem is and will remain difficult. We have put Lucite cones in the distal vessel at the point of the cut intima in an attempt to hold the intima against the wall. We are not doing that now. We have done it experimentally only. If the indications are followed we feel that this is unnecessary.

An aortogram is shown in Fig. 1*A*. There is some spillage at the point of injection, but there is excellent visualization of occlusion of lower aorta and both common iliacs.

The patient was treated by thrombo-endarterectomy and the postoperative aortogram is shown in Fig. 1*B*. This is one of the cases in which we failed to open the right common iliac. However, the cross-anastomosis from the reopened left internal iliac to the right internal iliac allowed this woman to walk a quarter of a mile on her bad leg, which is still the right leg, whereas formerly she could walk only 50 feet.

Figure 2 illustrates another case in which the lower aorta and both common iliacs were involved, treated by thrombo-endarterectomy. There was complete return of the distal pulses (Fig. 2*B*). Our longest follow-up is nearly three years, and none of the thrombo-endarterectomies has thrombosed subsequently

Ormand Julian (*Chicago*)

As Mr. Rob intimated, we have been more interested in the use of vein grafts in the femoral artery than artery grafts in the femoral artery. It seems to me there is very little difference in the ease with which these can be placed, that is, vein or artery, but whatever difference there is is in favor of using an artery.

The homologous vein has the disadvantages of a homologous artery in that it is not autogenous tissue, and an autogenous vein peculiarly is highly contractile. It goes into spasm, and until we learned how to use papaverine locally on these vessels we had great difficulty with that particular trouble.

The lasting properties of a vein, on the basis of probably an immature comparison between some of our results and those shown by Mr. Rob, seem to be superior. Of the 16 vein grafts initially successful, more than three years and less than four years at this time, in follow-up we have an average of only one subsequent closure of a graft. All 16 of these were reexamined slightly less than one year ago, at which time the four- and three-year follow-ups applied, and one had failed. This one subsequent failure was in an homologous vein graft In general, the grafts have remained patent during this entire period.

Figure 1 shows the preoperative arteriogram on the first case that was operated on in this series

We produced at that time what must be theoretically the most unfavorable situation that one can imagine. Except for the shortness of the graft, which of course is distinctly favorable, this dilatation was a source of great worry to us (Fig. 2)

For the first three years we did an arteriogram each year, and the caliber of the vein graft did not change (Fig 3) The recent follow-up did not include an arteriogram, but did show us that there was no change in the physical examination. So, this is a four-year survival of an autogenous vein graft, much too large in diameter



Fig. 1 Fig. 2.

Fig. 1 Preoperative arteriogram in autogenous venous graft.
Fig 2. Venous graft in place.

This tendency for vein grafts to remain patent over a long period of time is, I think, distinctly favorable, and encourages us to continue using vein grafts in distinction to homologous frozen arteries in the femoral system.

No such argument, of course, exists in the abdominal aorta. No one would think of putting a vein graft in the abdominal aorta unless some elaborate means were taken to prevent its dilatation. In aortic grafts so far followed there have been few failures We have one failure out of 50 major aortic grafts at all levels, and this failure might be of interest here.

A patient was operated on 20 months before death, for obliterative disease of the abdominal aorta. At the time of surgery we found unusual degrees of arteriosclerosis distal to the common iliac bifurcation, and in this early period we did not resect enough distally. As a result, postoperatively the patient did not have a restoration of pulsations on the right side. So, for 20 months this graft supplied only one leg. It atrophied, and when the patient died of acute pancreatitis after a bout of alcoholism, he exhibited an abdominal aorta of the size that would be necessary to supply just one leg.



Fig 3. Follow-up arteriogram on graft shown in Fig. 2.

I don't think we can call this a failure of the graft. We cannot indict homologous frozen grafts on this basis. It simply is an illustration that if one is to use these materials or any material in replacing the abdominal aorta in obliterative disease, he must remove enough aorta so that he gets a complete flow distally, because these tissues will atrophy. I should think that in a case of this kind we were lucky that it didn't thrombose completely because of the meager flow.

Tyge Sondergaard (*Aarhus, Denmark*)

I wish to report a case of aneurysm of the left coronary artery with an arteriovenous fistula to the right ventricle. The patient was an 11 year old

boy Congenital cardiac disease was diagnosed at the age of 5 He had been increasingly dyspneic on exertion, and often he had to rest while playing with other boys, but recovered quickly again. His mother believed she had had German measles during the pregnancy but no doctor was consulted—and she could not remember in what month.

The boy was frail, 10 kg below the normal weight for his age and 12 cm. too short. There was a right microphthalmus and reduced hearing in the left ear A thrill and a continuous murmur were found over the heart with one maximum in the second left interspace and one in the fourth space. P_2 was accentuated.

X-ray of the chest, lung function tests, and laboratory determinations were within normal limits

Cardiac catheterization disclosed a shunt to the right ventricle and to the pulmonary artery of approximately the same magnitude. The pressures in the right ventricle and pulmonary artery were just at the upper limit of normal. The preoperative diagnosis was aorticopulmonary fistula.

On November 25, 1954 the chest was opened through the bed of the left fifth rib An 8 mm. wide and 15 mm. long patent ductus was dissected out. The ductus entered the aorta higher up than normally, and the recurrent nerve was located 20 mm. further down on the arch of the aorta.

The ductus was clamped, but a thrill was still palpated over the pulmonary conus and through the pericardium an abnormal bulge could be seen.

The pericardium was opened just in front of and parallel to the phrenic nerve. A 60 mm. long, 20 mm. wide bright red pulsating mass partially covered with fat was now seen, the long axis approximately following the septum between the right and the left ventricles A strong thrill was felt over the mass. When pressure was exerted with a finger on the upper part just to the left and a little behind the pulmonary artery, the thrill disappeared and the mass collapsed. Pressure on the lower part caused a further bulge.

The diagnosis of an aneurysm on the left coronary artery with an arteriovenous fistula to the right ventricle was obvious.

The patent ductus was ligated doubly with heavy silk and a transfixion suture placed in between.

The 6 mm. wide coronary artery entering the aneurysm behind and to the left of the pulmonary artery was now dissected free and clamped, while the myocardium was observed and the electrocardiogram recorded continuously for ten minutes. The cardiac activity was unchanged and nothing abnormal could be noticed on the surface of the heart. The coronary artery was ligated doubly with silk tied over a piece of muscular tissue obtained from the chest wall. The wide entrance to the right ventricle was stitched. The remaining part of the aneurysm was now obliterated with three ligatures carried underneath the dilated vessel on a round curved needle and tied over a piece of gelatin sponge.

The thrill disappeared and no other abnormality could be detected.

The pericardium was closed partially, a drainage tube placed in the pleural cavity and the chest closed in layers

The postoperative course was uncomplicated. Three months postoperatively the boy was without symptoms. No murmurs were present, and the heart had decreased in size. On cardiac catheterization the pressure in the right ventricle was 20 mm. Hg and there was no evidence of any shunts.

Aneurysms on the coronary arteries are rare. They are classified as congenital and acquired—and out of more than 50 we have only found 7 with an arteriovenous communication. Four have been demonstrated at autopsy.

Sweet has reported a case in a 9 year old boy with a continuous murmur in the fourth right intercostal space. At operation a 6 mm. wide right coronary artery communicating with large tortuous veins which entered the coronary sinus was found. Sweet was afraid that ligation would cause myocardial infarction, and the chest was closed again.



Fig 1 AN. The aneurysm Some normal coronary vessels are seen just below PA The main stem of the pulmonary artery LPA. The left pulmonary artery AO The aorta with the vagus nerve. The black arrow is pointing to the ligated ductus.

Gross operated upon a 16 year old boy with a machinery murmur in the left fourth interspace. Six weeks previously the patient had had bacterial endocarditis. The preoperative diagnosis was patent ductus, but thoracotomy disclosed an aneurysm on the coronary artery (apparently similar to that in our patient) without a patent ductus. The malformation was considered inoperable and the chest closed.

Bjork and Crafoord have reported a 15 year old patient with a clinical picture identical with that of a patent ductus. At operation an aberrant branch of the left coronary artery communicating with the pulmonary artery was found and ligated, with cure of the patient.

Reich recommends surgical treatment in his book "The Uncommon Heart Diseases."

We found that temporary clamping of the feeding artery to the aneurysm in this case was without any influence on the action of the heart. Definitive treatment was judged to be possible—and it was.

J Maxwell Chamberlain (*New York*)

It may be hazardous to cross-clamp the aorta near the arch in old people because of the strain on the left ventricle and the cerebral circulation. This is especially true in the resection of high aortic aneurysms which have developed no significant collateral circulation.

A circulatory shunt avoids the hazards of proximal hypertension and is an immediate surgical substitute for collateral circulation. It provides nutrition to the distal vital organs and time for meticulous reconstructive surgery

I should like to describe a ten-hour operation on a 64 year old man, who had four aneurysms in the thoracic aorta The first was in the ascending aorta, one was immediately distal to the left subclavian, one was in the midthoracic area and the fourth was just above the diaphragm (Fig 1)

A heterologous frozen dried aorta, which had been washed and properly sterilized, was used as a shunt and was taken from a 225 lb pig It was anastomosed to the dome of the aneurysm in the ascending aorta at one end and to the abdominal aorta just below the diaphragm at the other end. When the shunt was working well the arch was cross-clamped between the left common carotid and the left subclavian in order to resect the three aneurysms and the entire descending aorta A homologous graft was used to replace the diseased descending aorta. The heterologous graft was then taken down and in the process the smallest aneurysm in the ascending aorta was resected by the tangential technique. The patient has made a good recovery without any evidence of paralysis The operation was performed in November 1954, at Fort Hamilton Veterans Hospital, Brooklyn, N Y

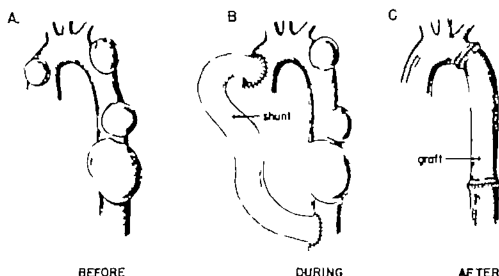


Fig. 1 Illustrating use of temporary heterologous shunt during resection of portion of thoracic aorta for multiple aneurysms

Ralph A. Deterling, Jr (*New York*)

Dr Cooley's observation of a lowered mortality rate among those patients with near normal or normal blood pressures, as compared to a hypertensive

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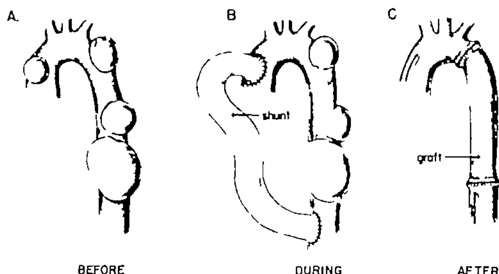


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Dr Cooley's observation of a lowered mortality rate among those patients with near normal or normal blood pressures, as compared to a hypertensive

group in which an aortic aneurysm was resected and a graft employed, calls to mind an experience which may be of related importance. Laborit visited our laboratory over a year ago and demonstrated his technique of hibernation therapy with a "lytic cocktail" composed of Phenergan, chlorpromazine and Demerol In the hope of protecting poor-risk patients of advanced age, with elevated blood pressure and significant renal disease, against the dangers of a lower nephron nephrosis following resection and grafting for aortic aneurysm, I employed his technique in 7 such patients. Subsequently I compared these patients with 7 patients of the same general age and blood pressure on whom I had performed the same operation. In the patients who received fractionated doses of the "lytic cocktail" during the hour or so preceding surgery, the blood pressure reached a plateau averaging about 115/80 mm. of mercury which was relatively easy to maintain throughout a four- or five-hour procedure, in contrast to an average blood pressure of 165/90 in the other group. Also, those given the drugs required far less anesthetic agent during operation than the others. At the time the aortic clamps were removed, the average fall in blood pressure was 27 mm. of mercury in the patients given the "cocktail," contrasted to the 135 mm. fall observed in the other group. In the latter, although the hypotension was of short duration, a pressor drug or additional blood administration was usually required. The amount of blood given during the operation was about twice that used in the treated group. These findings should be investigated by others as well

In regard to blood vessel replacements, I am certain that the panel will mention most points of outstanding interest During the past seven years our laboratory has investigated every conceivable type of vessel replacement including a variety of synthetic materials. Although there is no question that great care must be exercised in selecting or fashioning the proper size and shape of replacement and that strict vascular techniques should be employed, there is also no question that if the arteriosclerotic disease distal to the vessel replacement is advanced and severe, any graft has a good chance of failure.

We have recently published the results of a four and a half year study of aortic homografts in dogs and were distressed to note the marked thinning of the homograft following three years or so in the dog There was often a spotty destruction and even disappearance of the elastic framework of the original homograft in the late period. Although we observed no aneurysms in a series of 18 dogs, we must admit to this possibility at some subsequent time. We are continuing our long-term study with additional animals. In view of these observations, the difficulties in obtaining homograft material and the dilatation often encountered in the aorta of patients with aneurysms, we have come to favor the use of synthetic fabrics for the replacement of major vessels. It is of great importance that the surgeon exercise some intelligent judgment in his selection of synthetic material and type of fabric and that he know exactly the physicochemical characteristics of his material so that it may be reproducible when an additional supply is needed. We must interject a note of caution in the use of synthetic materials also, since certain synthetic fabrics obtained from casual sources contain dangerous impurities, and since the

performance in the vascular system of such grafts has not been studied sufficiently over an indefinite period of time. This would simply support our longstanding belief that blood vessel grafts have proven to be lifesaving for some patients but should not be used in any patient unless absolutely necessary for a satisfactory surgical procedure.

AN INTRODUCTION TO ATHEROSCLEROSIS (FOR SURGEONS)

IRVINE H. PAGE (*Cleveland*)

A lone internist among such an international gathering of surgeons is a strange anomaly. I can believe that I was invited only because occasionally a surgeon complains about the cloth provided him by the body and which he must sew. Surgical technique will be successful directly in proportion to the quality of the living tissues to be joined

The story I have to tell is not an ebullient one, it is the story of man's long fight with his environment. It is filled with doubts and uncertainties and, worst of all, there is little fact on which to build. It is the story of the struggle to keep vigilant, lest we stray from the path of truth and become hopelessly lost in a jungle of clinical impression and advertising promotion For those surgeons who feel that atherosclerosis is a subject with more than passing interest, I have presented it in more detail in a recent Lewis A Connor lecture.[1]

All subjects have their politico-economic aspects and atherosclerosis is no exception. It has taken about 4992 years for the medical profession to get busy with serious research on the nature of this disease. In the past ten years more and more money is becoming available for research, chiefly through the American Heart Association and the United States Public Health Service But most of the programs, including our own at the Cleveland Clinic, are woefully weak

ATHEROSCLEROSIS—A MULTIFACETED PROBLEM

The one thing, it seems to me, most likely to retard progress in the search for the cause of atherosclerosis is to formulate the problem from a too narrow point of view I am afraid that this is currently being done

I think you will find it helpful if the mechanism is visualized as a sort of water filtration plant in which lipid is being filtered through the vessel walls out into the tissue, with part of this being picked up and returned by way of lymph As in any filtering system, the pressure under which the filtration occurs is important, this is the blood pressure If it is preternaturally high, more filtration will occur, with the greater likelihood, although not the necessity, of augmented atherogenesis

If there are greater quantities of lipid in the blood, more lipid will filter through the wall and again there is a greater than average chance of some getting stuck in the wall and remaining there as a foreign body.

If the filter bed itself is abnormal, either as an hereditary trait or as a result of injury, more lipid may be held back at points of imperfection.

Besides these more mechanical factors, there are chemical ones The blood vessel wall has a slow but nonetheless real metabolism all its own. It may well even make and degrade cholesterol and other lipids found in atheromatous deposits Therefore the metabolic activity of the vessel walls must be considered as a facet of atherogenesis Further, the body itself and, more especially the liver, can synthesize cholesterol from a variety of precursors When cholesterol is reduced in the diet, the liver promptly responds by synthesizing more. The net effect on the total cholesterol content of the blood is just about zero It will be apparent, then, why a low cholesterol diet alone has no particular effect except for its nuisance value, which may be considerable.

LIPOPROTEINS AND ATHEROGENESIS

Lipid is the only major nutritional constituent which is insoluble in water The body must then solubilize it in some fashion. It does so by coating lipids with protein to form large molecular complexes known as lipoproteins Michel Machbouef was, I think, the first to recognize their importance. Although they were well known among specialists in lipid metabolism, Gofman brought them into prominence as regards atherosclerosis by introducing the ultracentrifugal method of determining them in the blood. This was an important and useful service. I think Gofman has erred in stressing too heavily the significance of small variations in lipoprotein content of blood in atherogenesis, just as others have erred in overstressing the importance of minor variations in cholesterol content of blood. Although abnormalities of either or both are doubtless of some importance, it seems unlikely that atherosclerosis would actually occur without many other facets of the mechanism of atherogenesis being present.

Lipoproteins are formed in the intestinal wall and liver, and are, very roughly, of two sorts First, the beta lipoproteins, which are heavily laden with lipid, and consequently are quite unstable. If they become stuck in the filter bed of the arterial wall, it is easy to see how they can break down, liberating large amounts of insoluble lipid. In this state, lipid acts as a foreign body with the influx or differentiation of macrophages and possible scarring of the wall. The alpha lipoproteins, on the other hand, carry less lipid and are correspondingly much more stable. Barr has especially stressed the importance for the body of keeping much of the blood lipid in the alpha form of lipoprotein. This is what women do and it doubtless contributes to their general durability as compared with men. Their estrogenic hormones seem to control, among other things, this ratio of beta to alpha lipoprotein. Androgens, unfortunately, have a contrary effect. Whether the sexual and atherogenic properties of these hormones can ever be separated, no one knows At the moment there are few males who care to become feminized for the sake of influencing one facet of atherogenesis Feminization may well influence other things as well.

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DIET AND ATHEROGENESIS

The amount of fat in the diet has a profound effect on the plasma lipids in some people and little, or none, in others. I have been constantly impressed by this fact in our hypertensive-nephritic patients. This situation makes the problem of the relationship of diet to plasma lipid level and ultimately to atherogenesis a very "difficult" one.

There is little doubt that some people take less fat in their diets than others and have lower levels of blood lipids. From this it is suggested by Keys that there is a direct and obligate relationship. This may be true, but it is not the only possible explanation. Secondly, it is assumed that if the blood lipid level is lower in some populations the amount of atherosclerosis should be less. And this turns out to be true. So the Bantus and the Japanese have much less atherosclerosis than Americans, Dutch, Danes and Swedes. Again, is this a cause and effect relationship? Possibly, but it must not be forgotten that heredity and environment may be even more important

About all that can be said with some assurance is that there may be a broad relationship between the total fat content of the diet and the level of blood lipid in many people, but not necessarily in all Further, this elevation, if sufficiently great, may be a factor in atherogenesis. From this it follows that the reduction of the fat content of our diet to roughly 15 to 20 per cent may be desirable I have done this myself and have achieved four things: (1) kept my weight down to a level where my clothes do not shrink, (2) slightly reduced the average cholesterol and lipoprotein content of my blood, (3) restored the bonds of friendship lost during an eleven months retreat on an almost fat-free diet, (4) put my arteries on notice that from now on they are on their own as I can think of nothing more that I care to do for them at this time. If it is worth reducing the fat content of the diet, it is worth keeping its total caloric content consistent with a youthful figure.

The contest between vegetable and animal fat remains unsettled. One still doesn't know which side one's bread is oleo'd on. Currently, the vegetable fat proponents seem to be gaining the edge, which will not please the dairy states But this is too big and too important a problem on which to pass snap or dividend-minded judgments. Another interesting but unexplored facet of diet is the problem of the effect manufacturers produce by processing of food, removing necessary elements which are present in the less highly refined food of the Oriental peoples.

OTHER METHODS OF PREVENTING ATHEROGENESIS

Most other anti-atherogenic methods presume that moderate lowering of plasma cholesterol will prevent atherogenesis. This may be true or it may be of small consequence among the other facets of atherosclerosis. This relationship we do not yet understand.

Iodides in various forms have an effect on atherogenesis in rabbits fed cholesterol; they prevent its deposition when they are fed in large doses. Does the same hold in human beings? If you think you know the answer, it is to be hoped that the method for the ante-mortem diagnosis of atheromatosis of

the aorta, cerebral and coronary vessels will not long be withheld. Clearly, the difficulty is that the effect of such drugs cannot be measured because of inability to measure the amount of atheroma present before and after the drug is given.

Plant sterols such as beta sitosterol, when given in large amounts, moderately reduce plasma cholesterol. This is an interesting demonstration but does not appear to be a very practical way to prevent arteriosclerosis

Female sex hormones have aroused considerable interest because of the all too recent observation that women are far better constructed than men. If the anti-atherogenic qualities of feminism could be preserved without the loss of man's pride and joy, a great victory would have been achieved for the longevity of men. Currently this seems unlikely; most men prefer to remain men even at the cost of atherosclerosis, which may or may not make sense.

Reasoning from the physiology of the circulation, I suggest that the failure to exercise is one of the ways to augment atherogenesis A blood vessel needs the massaging action of movement to keep lipids on their way through the walls Few other changes in life have been more remarkable than man's ingenuity in finding ways of not taking exercise. He has to take a taxi even to the next bar, which may be only around the corner It seems to me likely that this failure to exercise the blood vessels is an important contributing cause to atherogenesis

The effect of *weight gain and loss and obesity on atherogenesis is highly* contentious, owing again to the failure to recognize that no method, short of post mortem examination, is available to measure their effects Everyone agrees that from the statistical point of view fat people die earlier than lean. There is no agreement as to whether there is more atherosclerosis Weight *loss is usually associated with significant fall in plasma lipids and weight gain* with a rise. If no change in weight occurs and the calories are burned during exercise, even though they be large in number, plasma lipids tend to remain the same.

One word more, and this concerns commercial exploitation of alleged preventatives and cures of atherosclerosis This type of promotion could not, I think, be used if the name cancer were substituted for atherosclerosis. It is obviously easy to make claims for this or that vitamin or lipotrope because of the inability to measure the initial degree of atherosclerosis and the results of its administration. Recently, the claims have become nothing short of fantastic. It is a rich market since almost everyone beyond the age of 25 probably has some atherosclerosis What is being poured down the unsuspecting gullets of the American public in the name of prevention of vascular disease is both funny and shocking Even many physicians lend their names to the promotion of this sort of thing by publication of "clinical impressions of improvement after the taking of one or other of these nostrums and then suggesting the results are so encouraging that widespread clinical trial is immediately indicated. This is the old and slippery philosophy that the drug may not help the patient but it will do him no harm. It may not do him any bodily harm but it will shorten the stay of his dollars and gravely reduce the willingness of the prescribing physician to think clearly and critically about the patient's

problem. The problem of atherosclerosis will not be solved by conscience-removers sold in bottles; physicians must have their noses rubbed in it until the truth becomes established

It is seldom that an internist has a captive audience of surgeons and, believe me, I have enjoyed it. If I have sewn some doubt instead of stitches I will be satisfied.

REFERENCE

1 Page, I H The Lewis A Connor Memorial Lecture, Atherosclerosis An Introduction Circulation, *10*·1, 1954

PANEL DISCUSSION ON SUBSTITUTES FOR ARTERIAL SEGMENTS

CHARLES A HUFNAGEL (*Washington*), MODERATOR
ARTHUR BLAKEMORE (*New York*)
CHARLES ROB (*London*)
OSCAR CREECH, JR. (*Houston*)
HERBERT SLOAN (*Ann Arbor, Michigan*)
ERIC NANSON (*Saskatoon, Canada*)
D EMERICK SZILAGYI (*Detroit*)

DR. HUFNAGEL

In the course of the development of arterial substitution, it certainly is very interesting to me to see this large group assembled here for a discussion of cardiovascular subjects I can recall that around 1944 I approached Dr Gross, after a meeting of the Boston Medical Society, and told him that we had a series of animals in which we had replaced the aorta with frozen grafts He was extremely interested. However, following this it was almost five years before we could get anyone else who was sufficiently interested to accept this type of work.

We established a blood vessel bank, and for almost two years we could not get a patient. As we brought out problems of blood vessel substitution with plastic materials, this was similarly decried for a long period of time.

As time has gone on we have increased our armamentarium, and our experience is certainly bearing some fruit.

In order to facilitate matters here, I think we will briefly outline the various types of substitutes that we have available. The oldest, of course, are the simple blood grafts of homologous vein. Why this type of grafting was so long neglected, no one can explain.

The introduction of preserved arterial homografts came next in our modern era. The use of preserved vein grafts came at about the same time. It is obvious that one must have readily preserved material in order for these things to be available whenever one needs them.

The introduction of synthetics is more recent. The use of heterografts is again intermediate in this general development. Now the surgeon, instead of not having anything with which to replace the vessel, has the difficult task of making up his mind about what to use.

We would like to start off this panel by going over the problems of grafting with homologous grafts Each of the members of the panel will be given an opportunity to make a few remarks about some aspect that is particularly interesting to him.

MR. ROB

I should like to start by showing the method by which we store and bank our arteries. I would like to say first that we in this field have followed behind Dr. Hufnagel, and we are very grateful for the lead he has given us.

Our frozen artery bank was started in 1950. It runs at a temperature of −79° C. and is maintained in a deep-freeze with dry ice. We have modified the deep-freeze by the insertion of a cork lagged insulated container in which we place dry ice and the grafts (Figs. 1 and 2).

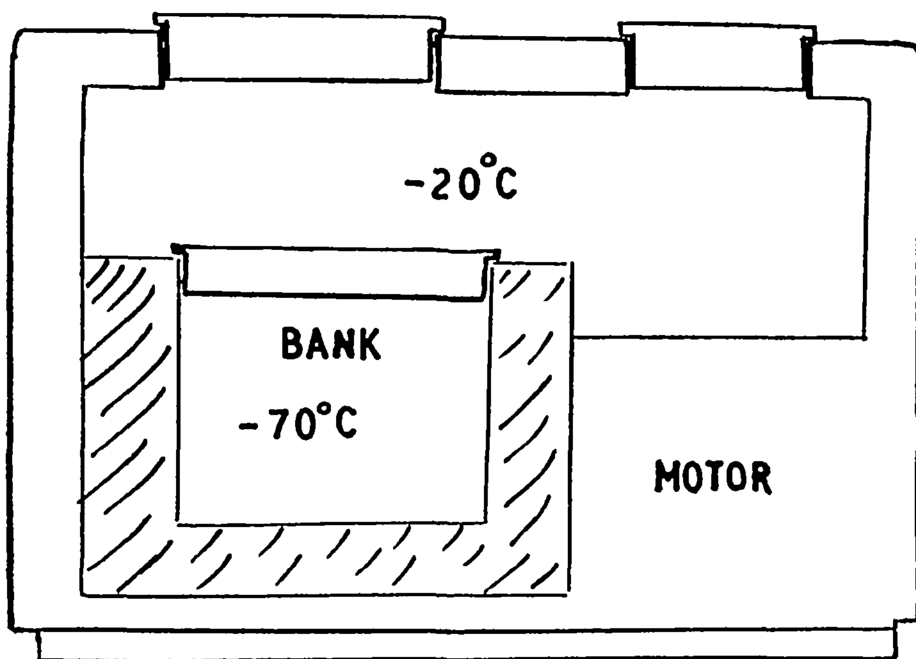


Fig 1 Diagram of modified deep-freeze utilized for artery bank.



Fig. 2. Storage of grafts surrounded by dry ice.

This bank worked extremely satisfactorily and still is very satisfactory We use this container as a method of temporary storage before we freeze-dry our present grafts

In 1953 we transferred our basic storage methods to freeze-drying The simple apparatus we use was a modification of the original penicillin freeze-drier used by Sir Alexander Fleming at our hospital (Fig. 3) The grafts are dried in small batches without external cooling We have achieved a water

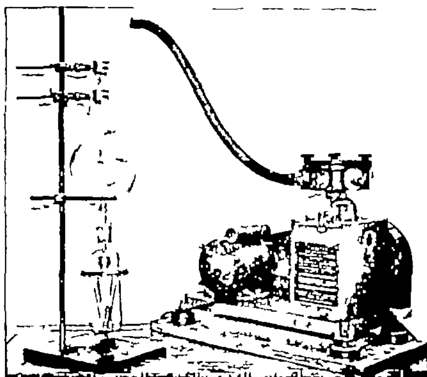


Fig. 3 Photograph of simple freeze-drying apparatus.

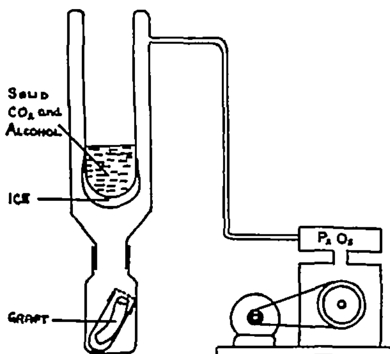


Fig. 4. Diagram of freeze-drying apparatus.

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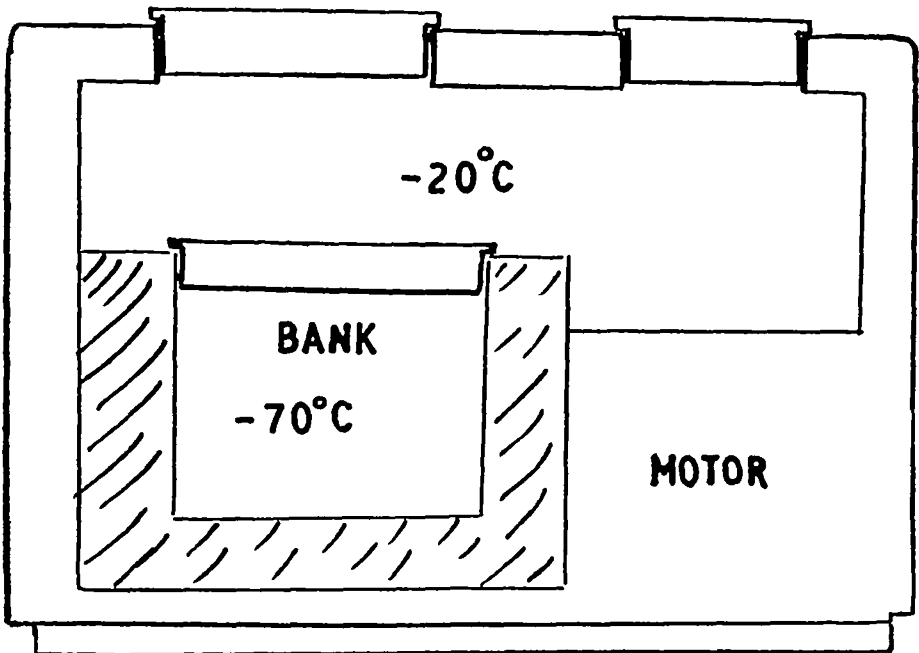


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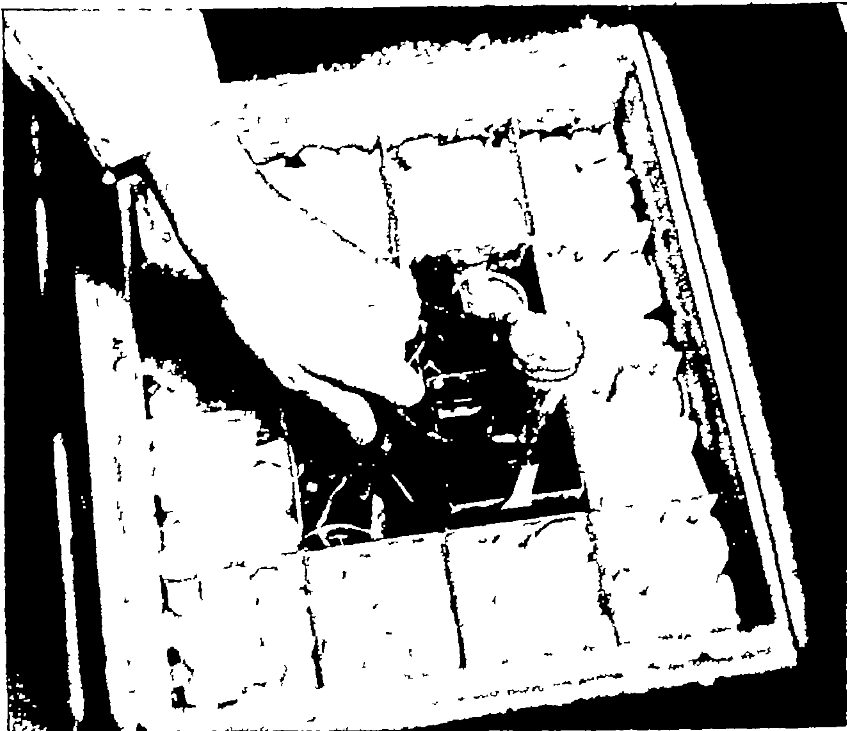


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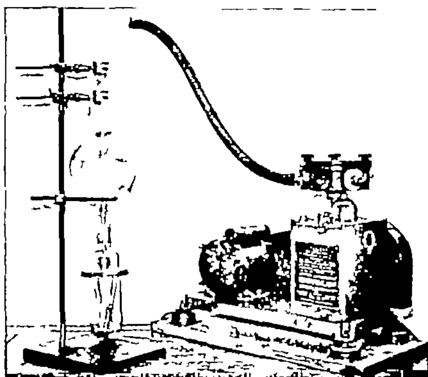


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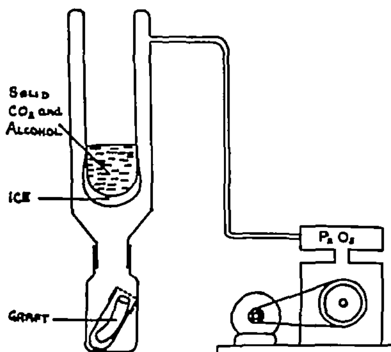


Fig. 4. Diagram of freeze-drying apparatus.

extraction of 99 2 per cent on the average, and the arteries histologically are satisfactory and almost indistinguishable from normal vessels.

The principle of our freeze-drying method is very simple (Fig. 4).

There is one other point I would like to raise, and that is the fate of an arterial transplant, because I think my views will differ perhaps from what some others might say.

Figure 5 shows a human aortic transplant removed five months after it had been inserted for a syphilitic aneurysm.



Fig 5 Autopsy specimen of aortic graft inserted five months previously for aneurysm of thoracic aorta

What about the fate of these arteries? We believe that in any arterial transplant which is more than 4 cm. long, the true endothelium of the intima grows out for only about 2 cm from the end, and that in the center of this transplant, where the lining looks satisfactory, it is not a true outgrowth of endothelium but is merely a blood clot with a flattened collagen surface derived probably from fibroblasts

As regards the replacement of the media and adventitia by fibrous tissue, this is a very much slower process in our view than some think, and we find that in a human transplant, removed and examined two years after insertion, there is still only a minimal amount of fibrous replacement of the media. The cells are there but they are dead, they look amorphous. On the other hand, the elastic fibers have persisted

DR CREECH

Early in our experience grafts preserved by refrigeration in a modified Tyrode's solution were used, but for the past two years freeze-dried homografts have been employed exclusively Most of the grafts are obtained from donors dying from accidental or homicidal causes. However, all patients coming to necropsy have their arteries examined, and if there is no gross evidence of arteriosclerosis the vessels are selected for homografts. Arteries are procured at necropsy usually within 24 hours after death, although when the

body was refrigerated promptly this time has been exceeded in some instances The entire aorta, common iliac, external iliac and femoral arteries are removed. For specific cases the external iliac and femoral arteries may be removed in continuity with the abdominal aorta and common iliac arteries, although generally these peripheral vessels are taken separately The aorta is divided just above the superior mesenteric branch into thoracic and abdominal portions for processing separately

Immediately after removal from the donor the homografts are sterilized with liquid ethylene oxide. We have found that the safest method of handling this toxic agent is to use it out-of-doors Since ethylene oxide boils at about 10° C., the tank must be kept in an inverted position in order to obtain the chemical in a liquid state The tubes containing the grafts are filled with ethylene oxide, and after 30 minutes the sterilizing solution is simply poured off onto the ground where it rapidly evaporates In this way the hazards of fire and inhalation of fumes are practically eliminated. After freezing at −70° C., the vessels may be lyophilized immediately or stored at −20° C. for freeze-drying at a later time.

As experience has accumulated, several changes have been made in the drying process (1) The oil diffusion pump, which was a constant source of trouble, has been eliminated and, although a lower vacuum is obtained and the drying time is increased, the vessels appear to be adequately dehydrated. (2) The dried grafts are now sealed in nitrogen rather than in a vacuum. This method obviates the need for expensive heavy-walled glass tubes, and thermal sealing of the tubes is more easily accomplished at atmospheric pressure. A tank of nitrogen is connected to the condenser with a fritted filter interposed (Fig 1) Upon completion of the drying process the trap and pump are eliminated from the system and nitrogen is allowed to flow into the condenser When the vacuum has been completely reduced, nitrogen begins to escape through the outlet port, and the tubes are sealed (Fig 2) More than 250 arterial homografts have been processed in this way with apparent satisfactory results

The changes occurring in homologous arterial tissue as a result of this method of sterilization and preservation have not been thoroughly investigated. Ethylene oxide produces no significant histologic change in the vessels, however, since this agent is a fat solvent, the lipid content of the homograft is reduced, and the tissue is somewhat dehydrated. Upon freezing at −70° C., numerous spaces appear between the elastic fibers in the media and are probably the result of the formation of ice crystals Elastic tissue stains, however, reveal no alteration in the elastic fibers It is probable that proteins are little affected, although recent studies indicate that the antigenicity of tissues may be altered by freeze-drying. When reconstituted in physiologic saline solution, the freeze-dried homograft appears narrower than normal and the spaces previously noted to follow freezing are still prominent. The endothelial layer is frequently absent, the subintimal zone is narrowed, and cell nuclei in the media are somewhat reduced in number

The changes that occur in human homografts following transplantation are similar to those observed in experimental homografts The freeze-dried vessel

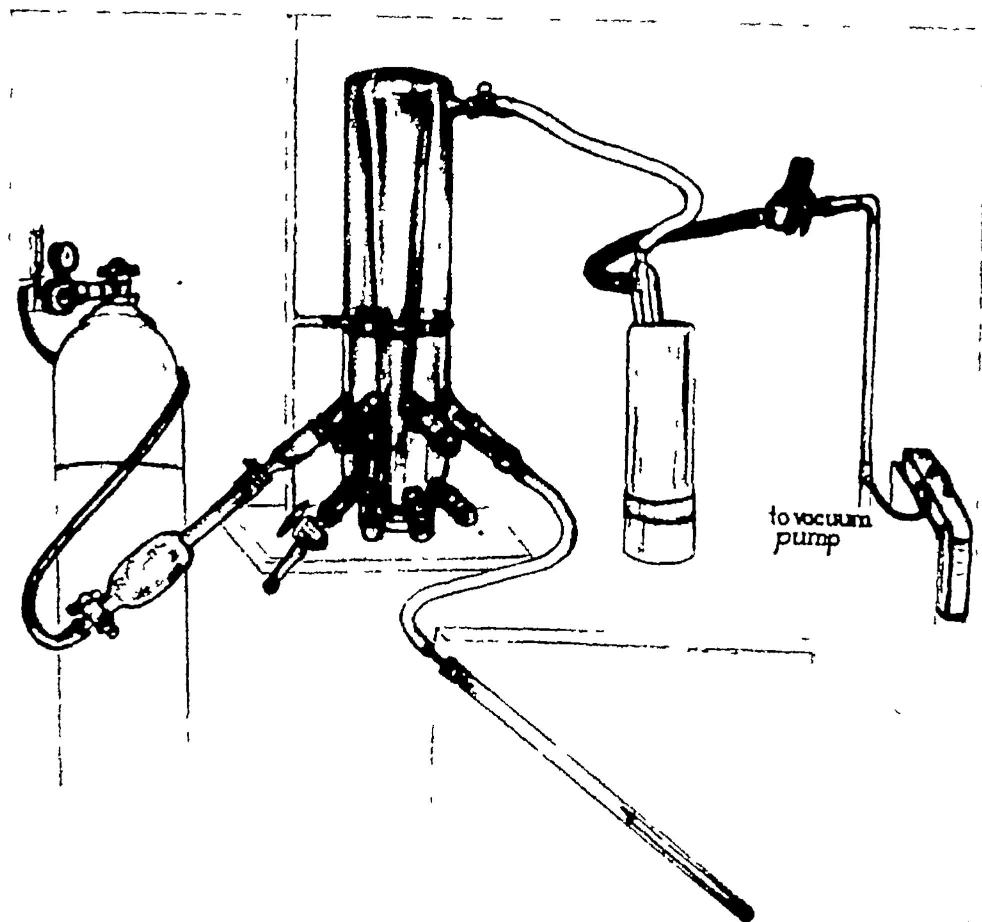


Fig 1. Sketch of freeze-drying apparatus showing the tank of nitrogen connected to the condenser with a fritted glass filter interposed Directly beneath the nitrogen inlet port is an escape valve which prevents the development of positive pressure within the system A plastic sphere is placed over the outlet port during the drying process and is held in place by the vacuum within the system After the introduction of nitrogen and reduction of the vacuum, the sphere falls away, allowing nitrogen to escape

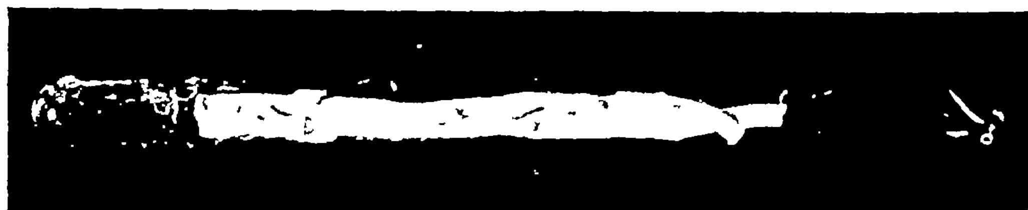


Fig. 2. Photograph of freeze-dried abdominal aortic bifurcation homograft

appears to lose its structural identity more rapidly than does the refrigerated homograft, but the process of deterioration and replacement by fibrous connective tissue of the host is basically the same (Fig. 3). In studying human homografts up to two years following transplantation, several interesting observations have been made. First, the inner surface of the homograft is not formed entirely by regeneration of endothelium from the ends of the homograft as has been observed in experimental studies In fact, only a small part of the inner surface of the graft is formed in this way, and then only near the anastomoses. In many places the innermost elastic fibrils of the media





Fig. 3 Photomicrographs of human aortic homografts, (*a*) 12 months, (*b*) 14 months, (*c*) 25 months following transplantation. In the 12 and 14 month old grafts the intima is thickened amorphous and relatively acellular; whereas in the 25 month old graft the inner surface is formed in part by the elastic fibers of the media and in part by a thin layer of fibrin. In each graft the elastic fibers are preserved, although in the 25 month old specimen evidences of fragmentation and disruption are present. A thickened hyalinized adventitia is noted in each graft.

are exposed, whereas elsewhere the inner surface may be formed by a thick, acellular, homogeneous layer somewhat resembling fibrin. Second, the elastic tissue of the media unquestionably is the principal supporting element of the homograft throughout the first five to six months following transplantation, and appears to remain unchanged for as long as one year. After this time, however, the elastic fibers begin to crack and fray and even become reduced in number Finally, we have noted the presence of hemorrhage within the walls of the transplants 12 to 25 months of age. Initially it was thought that these extravasations of erythrocytes occurred as a result of minute breaks in the inner surface of the graft. However, it has become apparent that this process probably results from rupture of newly formed capillaries in the granulation tissue which precedes the fibrous connective tissue replacement of the homograft. Thus, the formation of granulation tissue initiates hemorrhage which in turn results in formation of more granulation tissue. Although this is a natural mechanism by which the host adapts itself to homologous tissue, these intramural hemorrhages also may represent foci for the development of degenerative changes

DR. HUFNAGEL

As we have gone right to our very modern or current techniques, it might be worth while to review briefly the techniques that are available and that have been used

There are the various tissue culture methods in which solutions have been used at temperatures just above freezing, there have been the rapid freezing methods, and more lately the freeze-dried methods for the preservation of arteries. The freeze-dry methods can be used with grafts taken aseptically or sterilized by ethylene oxide, beta-propiolactone, and other methods That briefly summarizes the situation as it now exists.

In graft failures there is always a tendency to emphasize the ones which have not done well for very good reasons. One problem which is very common, and which is frequently overlooked, is that infection is extremely serious, whether it is in the suture line or immediately adjacent. Sometimes we assume an aseptic situation which is not necessarily present

DR. SZILAGYI

My assignment is to speak on that aspect of graft procurement concerned with sterilization of grafts. I believe there is general agreement that at present the most nearly ideal type of arterial substitute is the homograft. Prostheses made of plastic textiles have given great promise and some day perhaps they may even surpass homografts with respect to certain important qualities, but they are not as yet suited for general clinical use

About the only important shortcoming of homografts is the difficulty of obtaining them. This difficulty is the result of many factors, the most important of which is probably the time-consuming and cumbersome technique one must employ if one wishes to take the grafts aseptically Thus, a method of sterilizing grafts procured at routine autopsies without asepsis has been an urgent need. Several physical and chemical agents have been employed to

achieve sterilization of grafts, and at least one, the chemical compound ethylene oxide, has received a very extensive clinical trial Ethylene oxide has been used with success but its handling is cumbersome because of its qualities of volatility and explosiveness

During the past two years, benefiting from the extensive experiences of Hartman and LoGrippo (Am. J Clin. Path., *24* 339, 1954) in the sterilization of human blood plasma, and following the laboratory experiments of Trafas and Lam (Arch. Surg , *69* 415, 1954), we have utilized a method of chemical sterilization of arterial homografts with the organic compound beta-propiolactone. Beta propiolactone is a colorless, nonflammable and nonexplosive liquid with a flash point of 74° C. It is a surface irritant. In concentrations of from 0.3 to 1 0 per cent it has a very wide bactericidal, virucidal and fungicidal activity, without, at the same time, being deleterious to tissue proteins. Extensive experimental studies on human autopsy material have shown that beta propiolactone does not appreciably alter the elasticity and tensile strength of human arteries The histologic structure as well as the gross physical properties of the arteries treated with the compound in concentrations of 1 per cent or less is not affected.

On the basis of experimental studies, the concentration of 1 per cent by weight has been chosen for the technique of sterilization. The details of this technique are as follows

The arterial specimens are obtained in the autopsy room without aseptic precautions after the pathologist has removed all the thoracic and abdominal organs. The entire aorta is removed from the level of the ascending limb down to the bifurcation and beyond, as far as technically feasible to dissect. Whenever the permit allows, through special incision, the femoral arteries are also taken. It facilitates dissection and does not compromise the usefulness of the graft to transect the aorta at the level of the diaphragmatic hiatus The vessels are removed by a member of the artery bank staff because it is important to exercise caution in cutting the smaller arterial branches, these branches must be left with stumps of 2 or 3 mm. to make possible easy ligation just before use. It is of great importance to tie, by means of very heavy black silk left long, for identification by the undertaker the following branches innominate, common carotid, subclavian, internal iliac and external iliac arteries It is most desirable to cannulate with long plastic tubes, for the embalmer, the deep femoral and popliteal arteries when the common and superficial arteries are also taken.

The processing of the graft is carried out in three steps

1 After its removal the graft is washed in sterile saline and the saline wash is cultured. The graft is now taken to the blood vessel laboratory where the periarterial fat and areolar connective tissue are carefully trimmed away As formerly mentioned the stumps of the branches are not ligated until the time of use.

2. A familiarity with certain properties of beta propiolactone (BPL) will be useful at this point. Although quite stable in concentrated form, in watery solution at room temperature BPL rapidly hydrolyzes and loses its sterilizing effectiveness. Keeping low the temperature of the solutions to be described

presently until the actual beginning of the sterilizing process reduces this hydrolysis. In hydrolysis, acid breakdown products are formed; hence the need for careful control of the *p*H throughout.

The clean vessel is next transferred to a container (preferably a wide-mouth glass jar 8 x 16 cm., with a screw cap) kept in ice, in which has been placed a quantity of buffered normal saline.* The volume of buffered saline used depends on the size of the artery to be sterilized. Specimens no larger than the abdominal aorta with the common iliac arteries attached may be sterilized in a volume of fluid of 225 ml.; larger specimens require a volume of 450 ml. To the flask containing either 225 ml. or 450 ml. of buffered saline is now added 25 or 50 ml. respectively of 10 per cent BPL.† The addition of BPL solution is done slowly and under rigorous stirring, care being taken to bring the solution in contact with the entire inner surface of the jar and cap. The flask is now placed in a water bath for two hours at 37° C. From this time on the vessel must be handled aseptically.

3. At the end of two hours the graft is taken out of the flask under an ultraviolet hood, washed in a 0.2 molar phosphate buffer (7.4) and transferred to a storage flask. It is practical to use a Fenwal storage flask with a rubber stopper, which can be punctured with a needle to withdraw fluid for ascertaining sterility. The sterilized graft may be stored in any of a number of isotonic or nutrient solutions, or it can be lyophilized in precisely the same manner as a fresh graft.

During the past two years grafts processed with this method have been used in 59 grafting operations. The 59 grafts came from 37 donors. A wide variety of organisms were cultured from the grafts before sterilization but all cultures after sterilization were negative. The age of the donors varied from 12 to 61 years with a mean age of 34.7 years. The time elapsed between death of the donors and the time of autopsy varied from 1 to 22 hours, with a mean period of 8.2 hours. It is evident that the old criteria for acceptable grafts have been considerably relaxed. The only unalterable standard for the usefulness of a given graft is the state of its physical qualities as judged at the time of autopsy. Serviceable grafts can thus be obtained from people in their late 50's or early 60's. If a cadaver is well refrigerated, a lapse of time after death up to 24 hours will not cause significant enough post-mortem changes in the arteries to render them unusable. The cause of death of the donor has likewise lost much of its importance as a criterion of the usability of the graft.

As regards the results of the procedures in which grafts sterilized in the described manner were used, an over-all early patency of 84 per cent was obtained. The 59 operations included extensive aortic resections as well as

* The buffered saline solution is prepared by adding 1.68 gm. $NaHCO_3$ per 100 ml. of saline, or 4.2 gm. for 225 ml. and 8.4 gm. for 450 ml. of solution. For indicator phenol red is used (5 mg. per 100 ml. of saline).

† To make 10% BPL solution, add 2.2 ml. of concentrated BPL (sp. gr. 1.140) to 25 ml. of ice-cold water. This solution must be made fresh and kept col (on iced) until used. (Beta-propiolactone can be purchased from the B. F. Goodrich Company, Brecksville, Ohio.) A practical way to store BPL is to seal it in 2.2 ml. ampules (enough to make 25 ml. of 10% solution) and keep the ampules in the refrigerator.

Fig. 1 Histologic section of (upper) untreated and (lower) treated human arterial segment (aorta). (Hematoxylin and eosin stain, × 90)

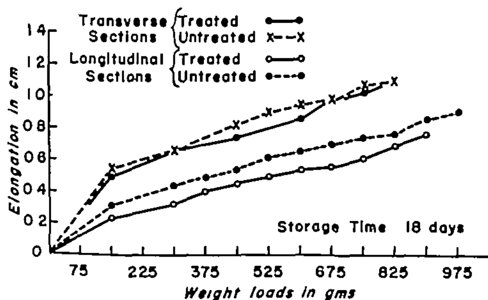


Fig. 2 Representative elongation curves of treated and untreated arterial segments (human aorta). The last plot on each curve designates the breakage point.

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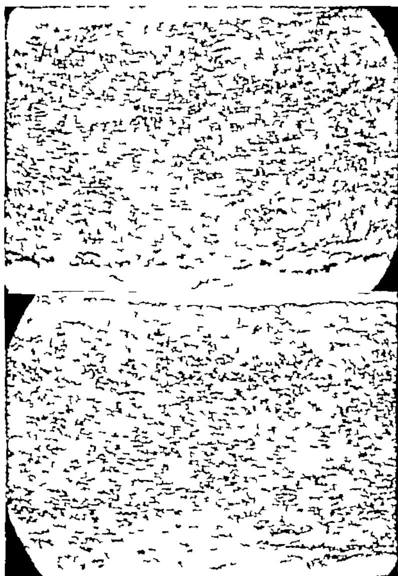


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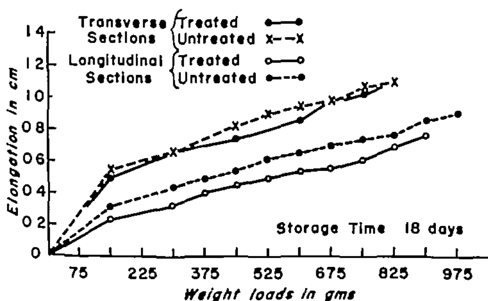


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replacements of long segments of femoral arteries (up to 40 cm.). No wound infections were observed in these cases.

On the basis of our experiences we may say that the sterilization of human arterial homografts with beta-propiolactone in the manner presented is a simple and effective method, and has made the procurement of such grafts much simpler.

DR. SLOAN

Like everyone else who has been interested in this field, we have not been able to get a sufficient supply of grafts to satisfy our needs.

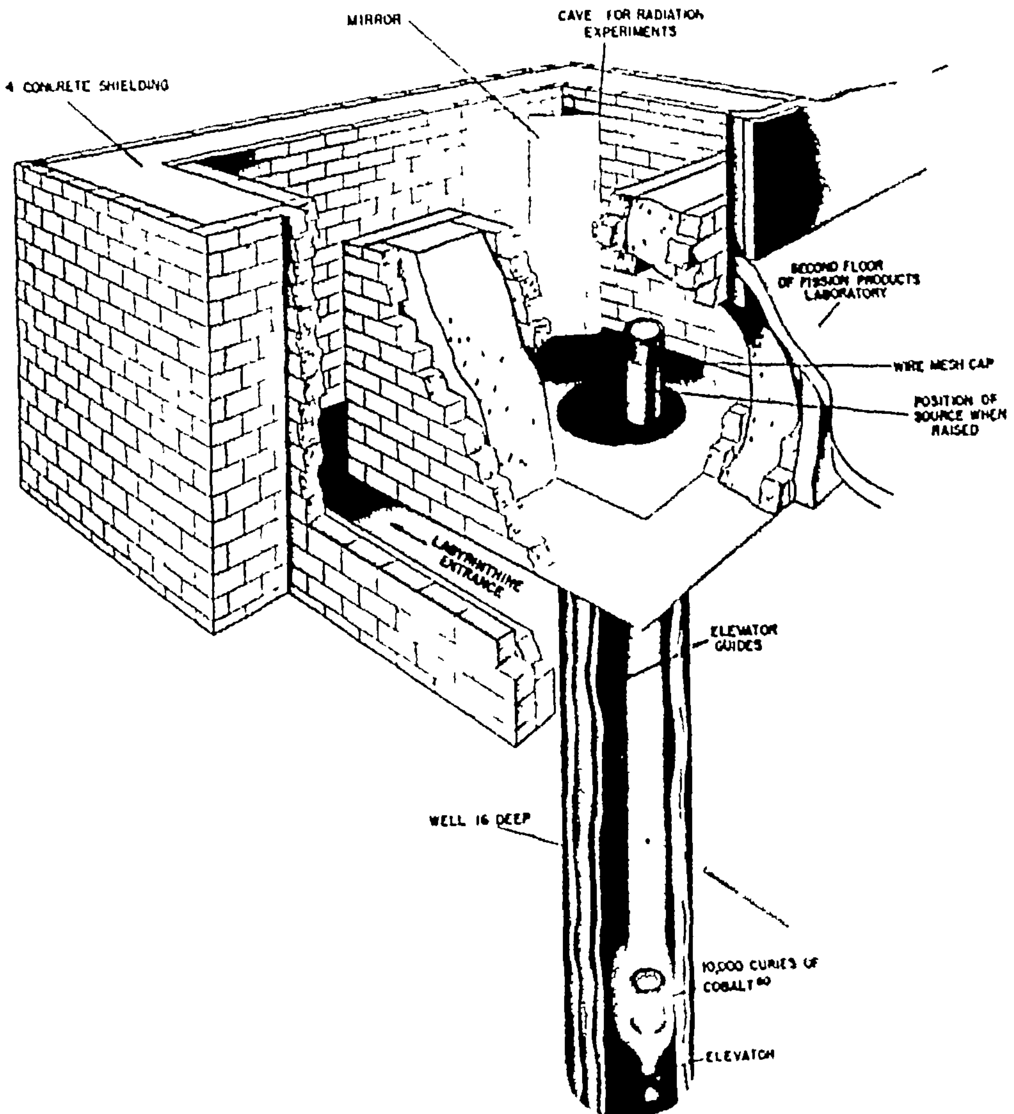


Fig 1 Diagram of the method of storing and handling the cobalt60 source.

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found that other workers on the University of Michigan campus were employing a 10,000 curie course of gamma radiation, cobalt^{60}, to preserve foods It became quickly apparent from the work that was done that gamma radiation is a beautiful way to preserve such things as meat and potatoes, so why not try it on aortic grafts?

We carried out a series of dog transplants after various amounts of radiation, and found that the grafts closely paralleled those that had been simply frozen-dried.

Figure 1 illustrates the radiation source, with the bomb at the bottom of a 14 ft. well of water The substances to be radiated are placed about the bomb in the upper room, and the bomb is pulled up into the room and radiation is carried out.

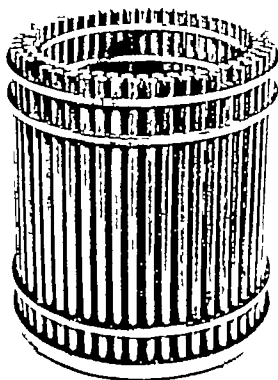


Fig. 2. The cobalt^{60} source Homografts to be sterilized are placed in the center of the source.

A study of the contaminants which we found in our own grafts suggested that 2 million r e.p would sterilize the grafts 'R.e.p " is roentgen equivalent physical," which is the scientists way of measuring radiation.

Figure 2 shows the actual radiation source. We are very fortunate because we can put our grafts right down in the middle of this radiation source, and they remain there for about 8 hours

A section of dog aorta is illustrated in Fig 3 An irradiated graft has been implanted and has remained in place for over six months. I think the majority of our microscopic sections parallel those you have already seen today A rather limited clinical experience of some 12 cases of human material suggests that these irradiated grafts are just as satisfactory as any that have been prepared.

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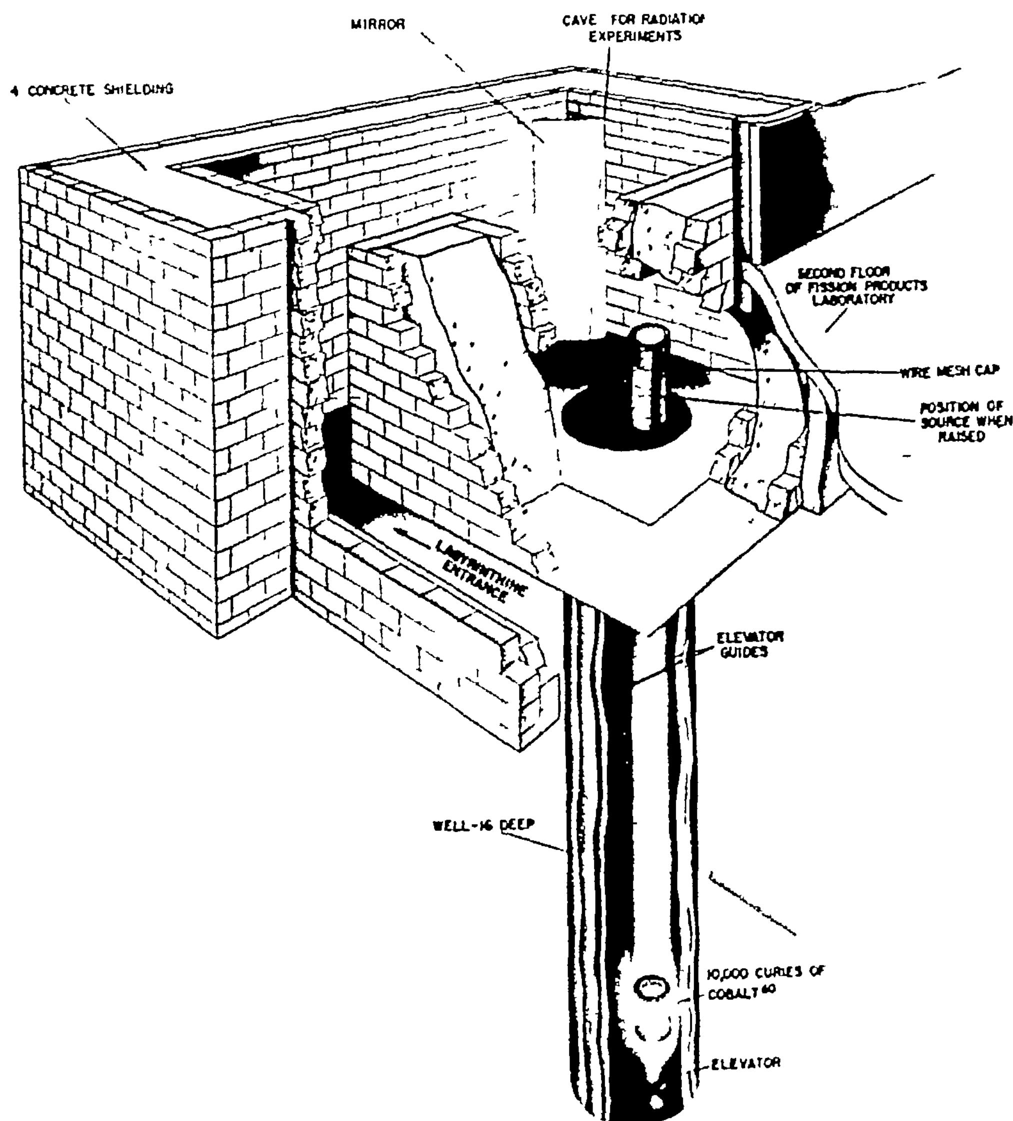


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In an attempt to do this we turned to a different form of sterilization from the one Dr Szilagyi has employed. Gamma radiation is a very excellent means of destroying bacteria; as a matter of fact, gamma radiation will kill everything from viruses to yeasts if the dosage is sufficient

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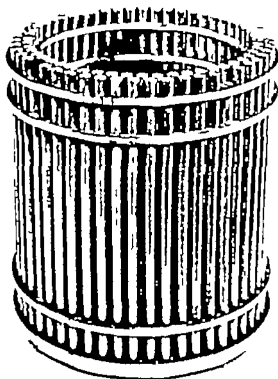


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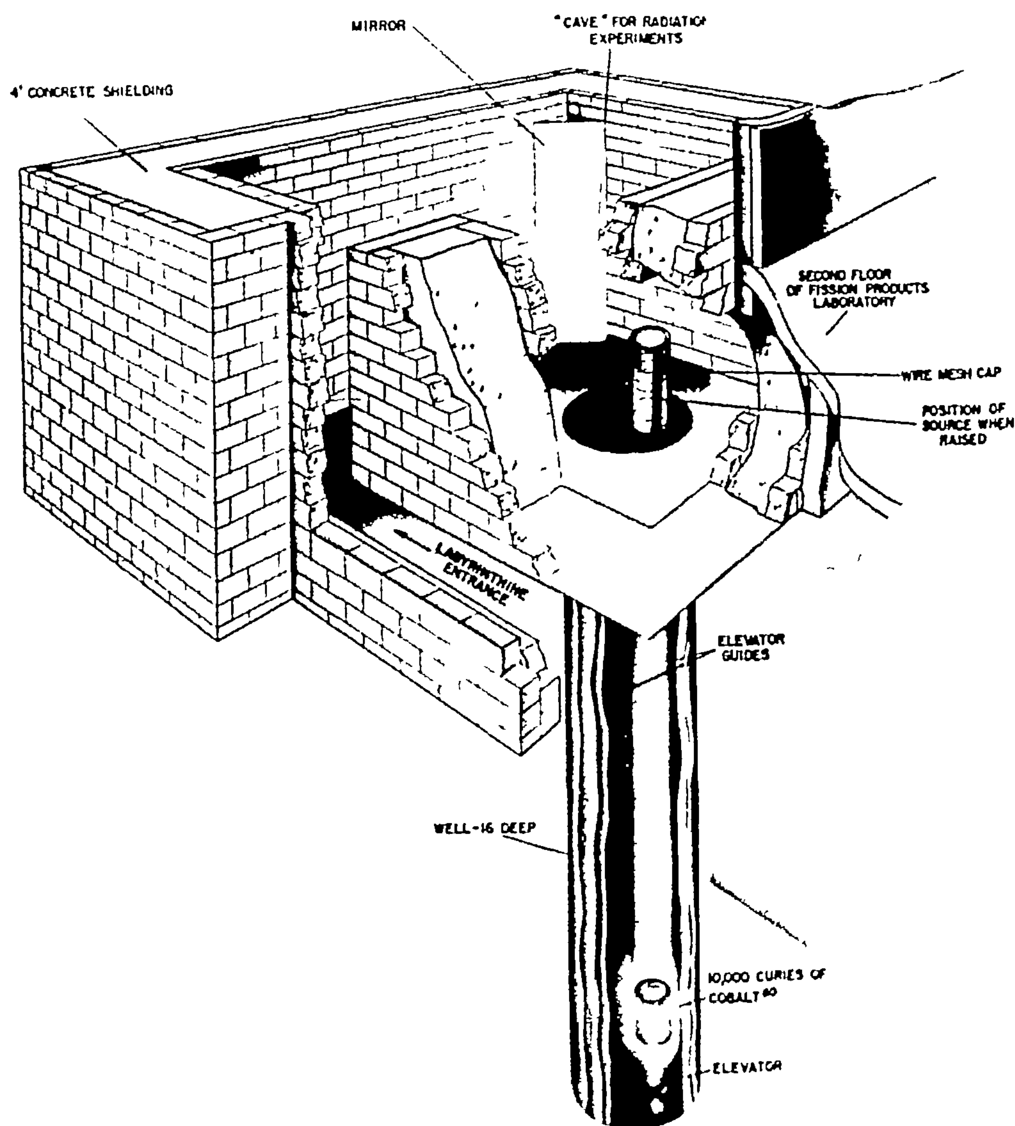


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who has pioneered in this effort, might give us some of his thoughts about the matter now

DR. BLAKEMORE

This reminds me of the early days when Jerry Lord and I, working on some quick method to get vessels together and to preserve them, did quick-freeze veins for grafting purposes

In the meantime, we were beginning to get some ruptured arteriosclerotic aneurysms of the abdominal aorta. We were looking around in the middle of the night for something to put in, and we stumbled on some of the straight tubes. I think the first replacement we tried was with a stiff plastic tube. There were other difficulties at the time which Dr Hufnagel had not quite settled, such as the multiple point method of fixation of these rigid tubes.

About five years ago my associate, Dr Voorhees, was doing some valve replacements in dogs, and came up with a specimen six weeks old, in which a silk chorda tendineae (in fact, two of them) had been placed in the left ventricle.

Upon examination it was perfectly obvious that these silk chordae tendineae looked quite like the rest of them, being covered by endocardium. Dr Voorhees came up with the idea that perhaps if you could take a plastic weave of some kind and shape it into a prosthesis, having the proper sort of weave, you could keep it from leaking blood, and perhaps the blood might go through and the tissues might grow through from the outside and eventually participate in keeping such a prosthesis from clotting.

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We started using Vinyon N because it was available. This cloth was fashioned into tubes and was used primarily in the aorta of dogs The feeling was that if this succeeded we could have a more physiologic type of prosthesis.

Our experience to date has extended over a five-year period, and encompasses data from 70 experimental animals and 44 human cases.

Initially this Vinyon N cloth was shaped into tubes, cut slightly bell-shaped at the end, and cuffed back to facilitate adjustment for length (Fig 1) We found after one accident that the single seam which caused one failure proved we should put in a double stitch line in the seam.

Figure 2 shows the Vinyon N cloth prosthesis in place in the aorta. You can see the vena cava to one side. This is after hemorrhage had ceased. There is always a transitory period of leaking which is not particularly serious in dogs, and it diminishes after 20 seconds and ceases after 30 seconds

Figure 3 shows an opened prosthesis, removed two years after implantation. You will note between the two suture lines the type of covering which has been best described by us as a functional intima. It is primarily due to fibroblasts growing through the meshwork and flattening out. We realize that the intimal growth from the two ends of the vessel bridge is not extensive.

Figure 5 shows a three-month-old prosthesis employing a braid weave of Orlon, not a flat weave. There is some growth of intima down from each suture

I admit that many workers do not have a source of cobalt[60] with which to irradiate grafts, but I believe there are other satisfactory means of sterilizing such grafts, and I think the problem of obtaining a sufficient supply of homografts has now been solved.



Fig 3. Irradiated, lyophilized segment of dog aorta examined 20½ weeks after insertion

DR. HUFNAGEL

It is quite apparent that the problems of obtaining, sterilizing and processing homografts for use are not small ones. It entails either a considerable apparatus or personnel to operate the various processes.

One has to have some temperature control, initially at least, although the freeze-dried material, once it has been processed, is then stored at room temperature.

I think it is worth while to point out that some of the older methods, such as rapid freezing, actually were not bad, although they have not been so widely used recently. They are perfectly satisfactory methods, but because of their inconvenience, they have been replaced by the newer processes. I think it should be pointed out, too, that there may be other advantages in terms of freeze-drying which are not necessarily present with grafts kept at freezing temperatures or at temperatures slightly above freezing. These have not been entirely established, however.

Since all of this has occurred, our own experience in the last three years has been with freeze-drying alone, and for the last two and a half years, all grafts have been taken without aseptic precautions. We have not had any difficulties that have been related either to the processing or to the sterilization. In this experience, we have dealt with more than 150 grafts.

The problem as it now exists in all of our minds, I am sure, could be simplified if we had a more readily available source of material. Dr. Blakemore,

who has pioneered in this effort, might give us some of his thoughts about the matter now

DR. BLAKEMORE

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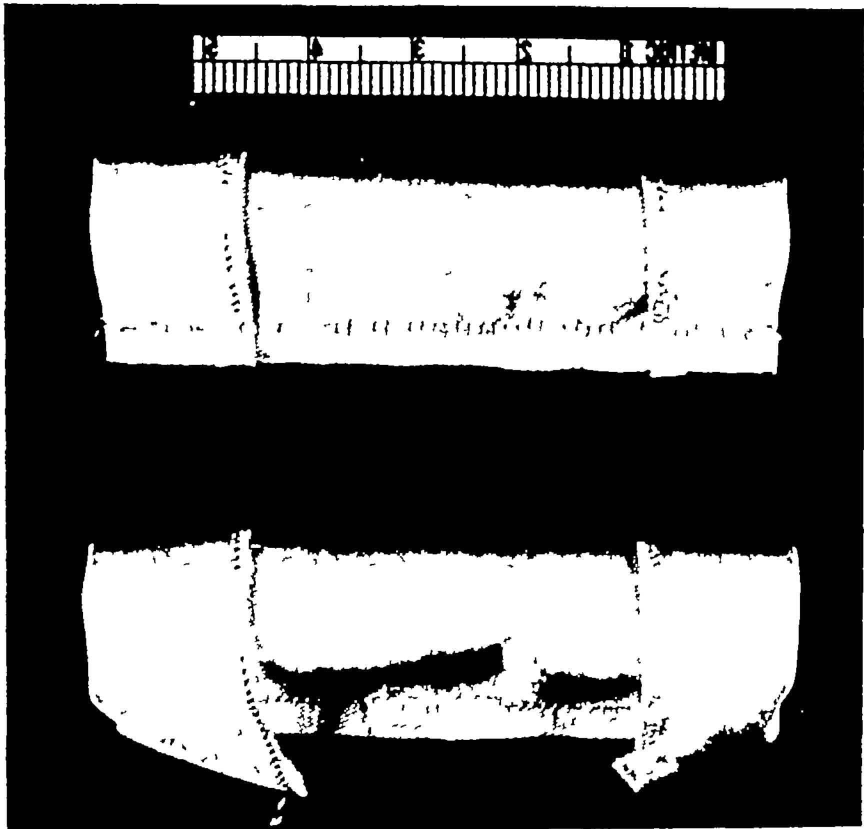


Fig 1 Tubes of Vinyon N cloth The ends of the lower tube were bell-shaped before being cuffed back

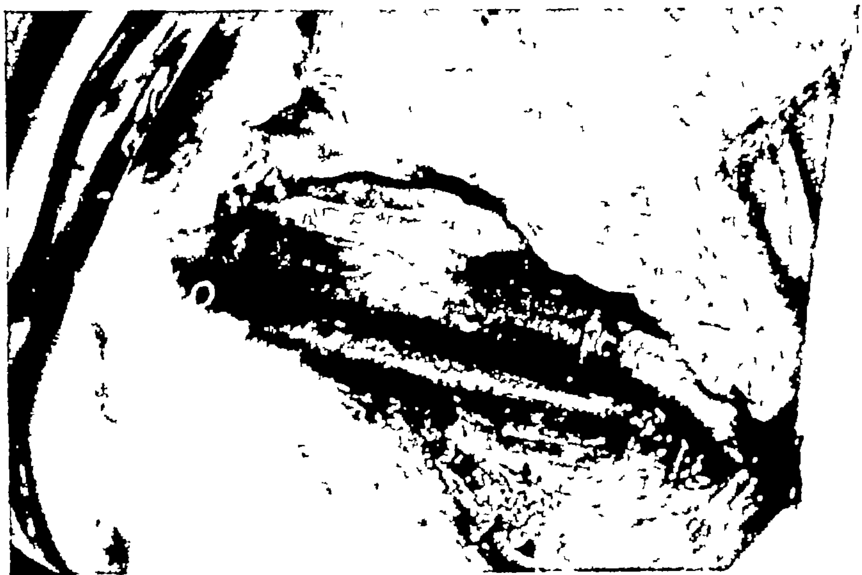


Fig 2 Appearance of prosthesis immediately after implantation Hemorrhage has ceased following clotting in the interstices of the cloth The vena cava is adjacent to the prosthesis

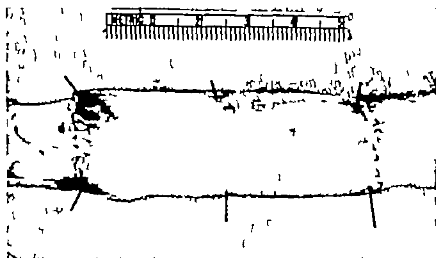


Fig. 3 Prosthesis removed after two years of implantation. Note almost perfect "intimal" lining.

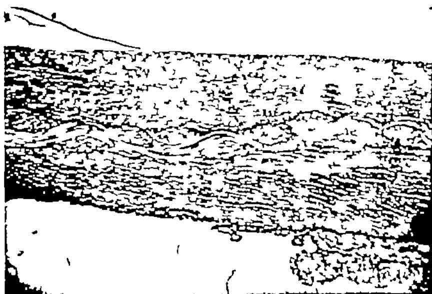


Fig. 4. Microscopic section of prosthetic substitute shown in Fig. 3 In the center are the fibers of the Vinyon N cloth. Above is the functional intima. Below is a small amount of fibrous tissue covering the prosthesis.



Fig. 5 Orlon prosthesis three months after implantation. Note the incomplete development of the intima

line, but the central area is not at all covered with so-called functional intima.

Figure 6 shows a bifurcated, braided tube which is of proper design. We realize that there are certain advantages in the braided tube. The main one is the absence of a seam. Another is that it has a certain amount of adjustability so that if you err in the estimation of the gap you are going to bridge, the braid compensates very nicely. This is in contrast to the flat weave which has little or no elasticity.

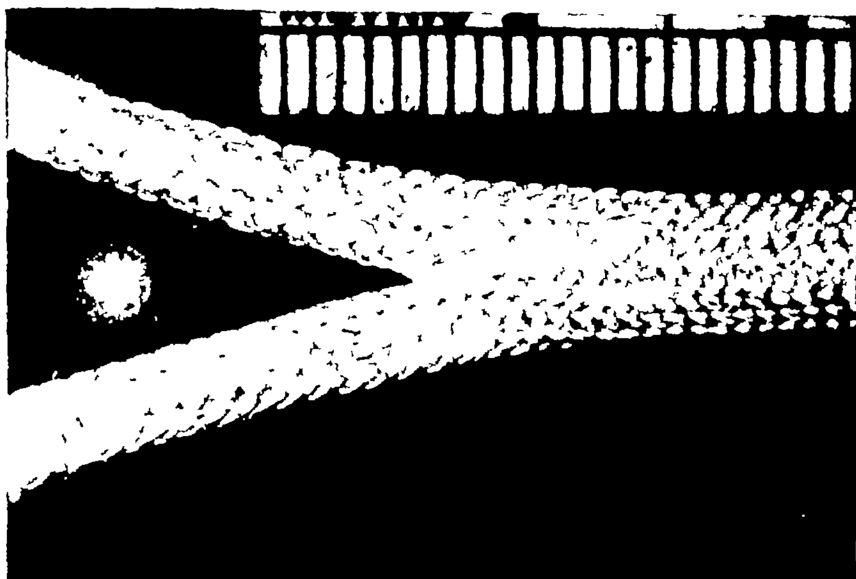


Fig. 6. A seamless braided "Y" prosthesis.

DR. HUFNAGEL

It might be worth while to mention here that one of the things one might have expected is clotting in any such prosthesis. It was pointed out a long time ago that in certain types of materials, blood surface contact depends upon water repellency and capillary attraction, and a great many studies have been carried out on the use of materials like methyl methacrylate. It has been well shown that the clotting time of blood in contact with this surface is greatly prolonged over what it would be in contact with other foreign surfaces like glass or certain metals. This property, which probably had best be termed hemorepellency, does bear some relation to the coagulation of blood in tubes of this sort. Orlon is another material in the methacrylate series apparently having this property.

Water absorption in plastics is often a problem, because in some cases there appears to be an effect of materials used in plasticizing or stabilizing such compounds and polymers. Materials which absorb water over a long period of time may differ from the relatively inert polymer itself, and in this way set up reactions which are quite undesirable. This is a large subject in itself, and I would merely like to call it to your attention. Just any plastic is not necessarily desirable.

In relation to braids and flat weaves, I think Dr. Blakemore might comment about the relationship of growth of covering of endothelial-like materials in relation to the type of weave, because I think there is some relation.

Dr Nanson, you have been interested in nylon, particularly nylon taffeta. Would you like to speak?

DR. NANSON

I became interested when I first saw Dr Hufnagel using Orlon for grafts He seemed to have a corner on the Orlon, so we had to look for other materials. Dr Bahnson suggested to me that perhaps we might try nylon, so we went to the shops and got some nylon taffeta, proceeded to try it out, and found it worked very well.

The advantage of the nylon taffeta is that it is readily procurable. It is generally of a standard weave which does not leak.

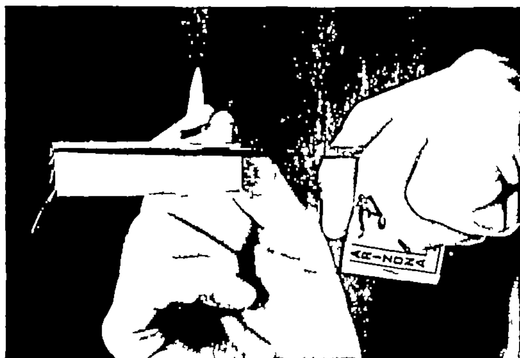


Fig. 1. Method of fusing nylon taffeta fabric into a tube using a Bainbridge clamp and a match.

I am not very good at sewing, and I didn't much like the idea of the cuff turned back. I thought it might be more difficult to suture into the vessels A friend who was a worker in plastics said, "Why don t you melt it and let it fuse?"

In essence, to make a suitable tube of nylon taffeta all that is required is a pair of clamps and some matches One holds the lighted match to the material, and the nylon melts and runs back upon itself (Fig 1) It is reasonably strong at the ends and actually one has to use rather strong force before it can be broken. To seal the ends, one runs the flame around the end. Again, the nylon is melted and flows back on itself, giving a nice edge but without the necessity of turning back a cuff. The sutures can be inserted quite close to the edge.

Figure 2 shows a tube taken from a pig one month after it was put in. It was perfectly evident that the endothelium in the pig grows at a far greater

rate than in the dog. We must remember that most of the experimental work has been done on dogs.

Figure 3 shows a nylon taffeta prosthesis in the aorta of a dog removed at the end of four months. Low and high power photomicrographs show the cellular changes which have taken place around the tube (Figs. 4, 5 and 6).



Fig 2 Nylon taffeta "graft" after implantation for one month in a pig Note that the fabric is almost completely covered with smooth endothelium

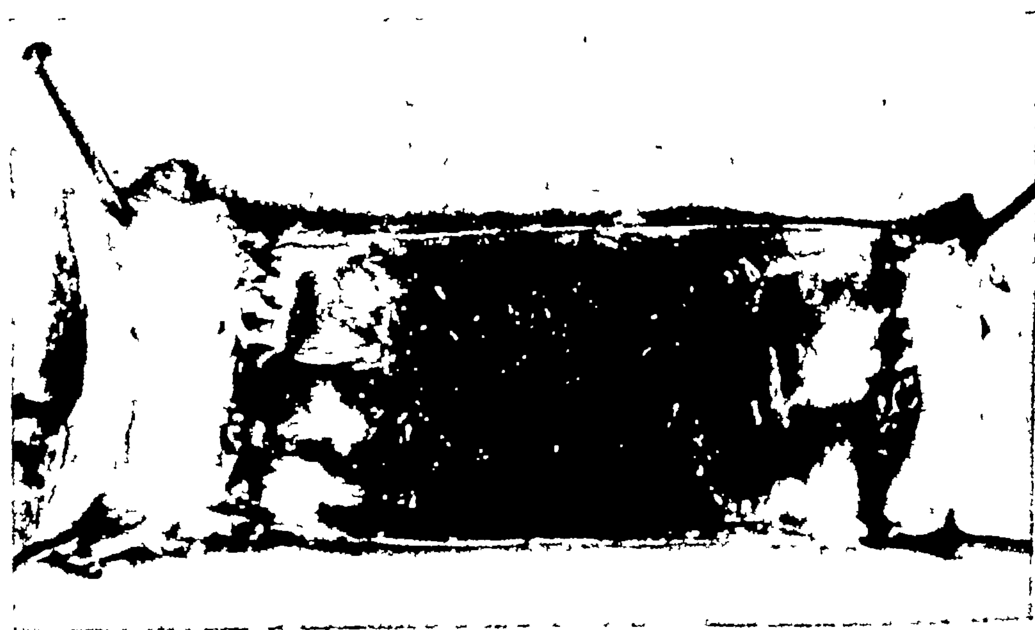


Fig 3. Nylon taffeta "graft" removed after implantation of four months in the aorta of a dog Endothelialization is incomplete

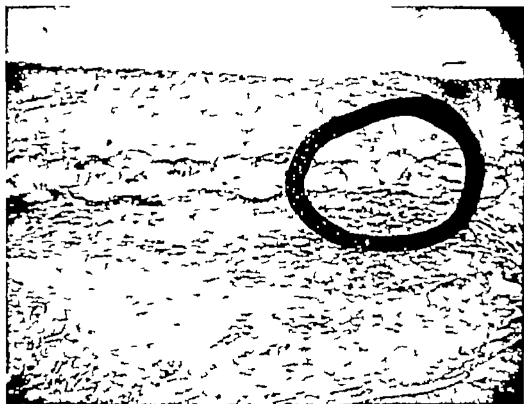


Fig. 4. Low power microscopic section of specimen shown in Fig 3 The fibers of the nylon are easily seen. The smooth intimal surface is above.

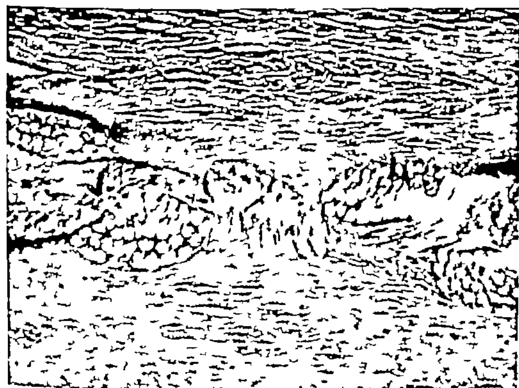


Fig. 5 Higher power magnification of area circled in Fig. 4. Note the fibrous tissue bridges passing through the interstices of the fabric.



Fig 6. High power photomicrograph of prosthesis removed 21 days after implantation into aorta of dog. The invasion of the fabric by fibroblasts is well shown.

DR. CREECH

A variety of synthetic textiles have been employed as arterial substitutes. The one with which we have had the most experience is made of Orlon cloth of 90 x 100 mesh and 100 denier. The prosthesis is fashioned on the sewing machine (Fig. 1) and is then placed on a mandril and its external surface coated with a thin layer of liquid vinyl plastic. The plastic dries in about 24 hours, leaving a flexible impervious prosthesis (Fig. 1*b*). These Orlon substitutes have functioned satisfactorily in 12 clinical cases (Fig. 1*c*).

A knitted, seamless tube of Orlon has also been employed clinically with satisfactory results. This prosthesis was developed by Dr. Paul Sanger at the Experimental Textile School of the University of North Carolina. The knitting process is accomplished with 80 needles to the square inch, and with two threads of Orlon (200 and 150 denier respectively) per needle. The tubes for aortic and common iliac replacement are knitted separately, then sewn into a bifurcation prosthesis (Fig. 2*a*). The resulting branched tube has an outer ridge where the three components are joined, but the inner surface is virtually seamless. This prosthesis has some lateral stretch and is relatively easily sewn to the host vessels. Although momentary brisk bleeding occurs following release of the occluding clamps, clotting takes place within a minute (Fig. 2*b*).

Braided nylon tubes have been used experimentally (Fig. 3). These tubes are constructed on a commercial braiding machine and are formed around a soft rubber mandril. The prosthesis has a weave of 90 x 44 strands per square inch When the tube has been woven it is dipped into liquid vinyl plastic in order to hold the threads together, and when the plastic is dry the mandril

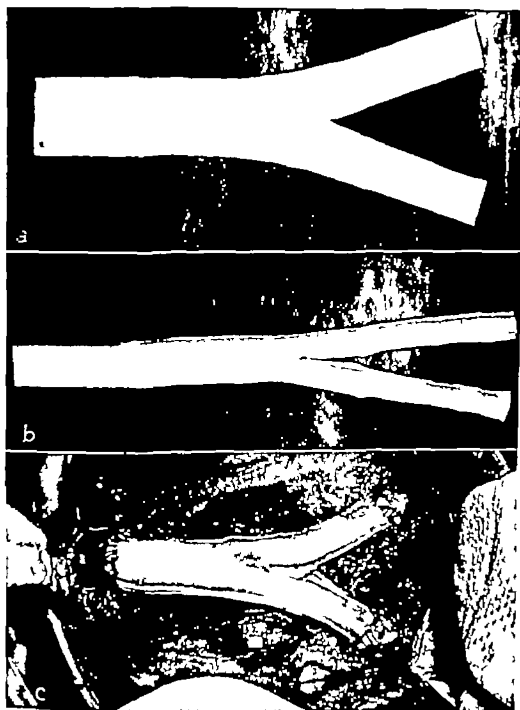
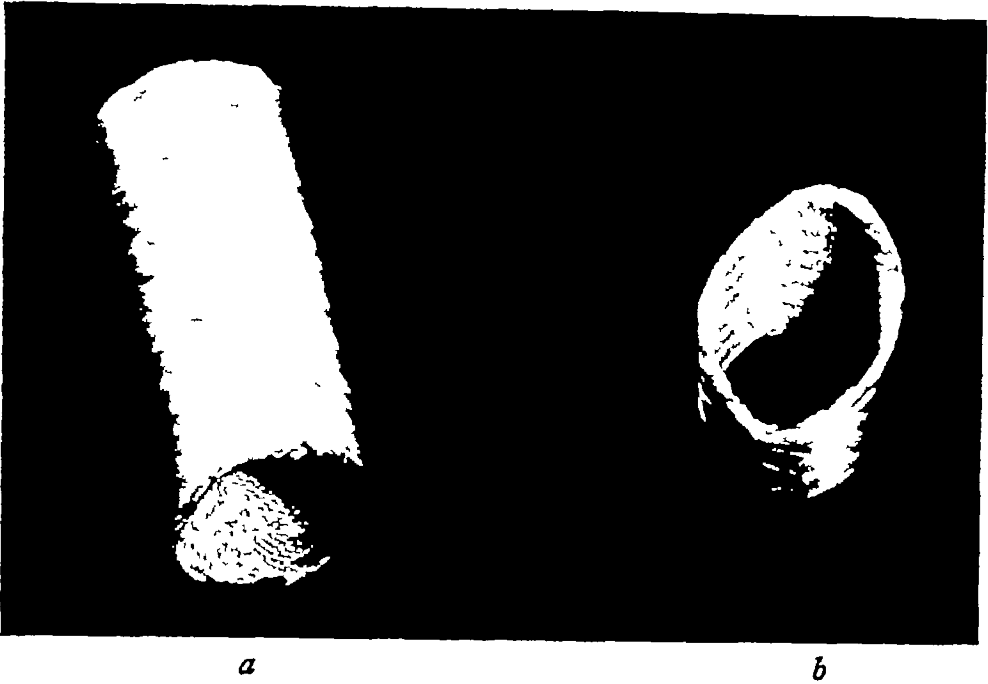


Fig. 1 Orlon cloth prosthesis (*a*) before and (*b*) after coating with liquid vinyl plastic. The resulting tube is pliable yet retains its shape. (*c*) Photograph of this Orlon prosthesis which has been implanted into the abdominal aorta following resection of an aneurysm.



a *b*

Fig. 2. A knitted Orlon prosthesis (*a*) before and (*b*) after implantation into the abdominal aorta

Fig 3 *a*, Photograph of seamless, braided nylon tube The outer surface has been coated with liquid vinyl plastic *b*, The tube in place.

is removed. The resulting tube has a smooth outer surface and a rough inner surface formed by the interstices of the thread. When implanted into the thoracic aorta of dogs, these prostheses function satisfactorily They are encysted by adjacent host tissues, and a fibrin network is laid down in the interstices of the thread to form a smooth inner layer which ultimately becomes organized. We have been unable, however, to construct a satisfactory bifurcation prosthesis from this material.

In summary, about 250 arterial substitutes have been employed clinically, 203 of which were implanted into the aorta and the remainder into peripheral vessels. The early results with the aortic substitutes are as follows There were 30 thoracic aortic homografts used, 3 of which were preserved by refrigeration in a nutrient solution and 27 by freeze-drying Among this group there was one early graft failure. Of 158 homografts implanted into the abdominal aorta, 13 were preserved by refrigeration, among which there was one early graft failure, and 145 were preserved by freeze-drying, with one graft failure. Twelve impervious aortic bifurcation prostheses made from Orlon cloth have been used, and 3 pervious knitted Orlon prostheses have been implanted. No early failures occurred. Thus, of 16 homografts preserved by refrigeration in balanced salt solution there were two graft failures, and in 145 freeze-dried homografts there was one graft failure. It is, of course, too early to determine the late results with these types of substitutes Unquestionably, degenerative changes do develop in homografts irrespective of the method of preservation, but whether these changes will significantly affect function remains to be determined It appears likely, however, that a suitable vascular prosthesis constructed of synthetic materials will be developed and will replace the arterial homograft as a vascular substitute.

DR. HUFNAGEL

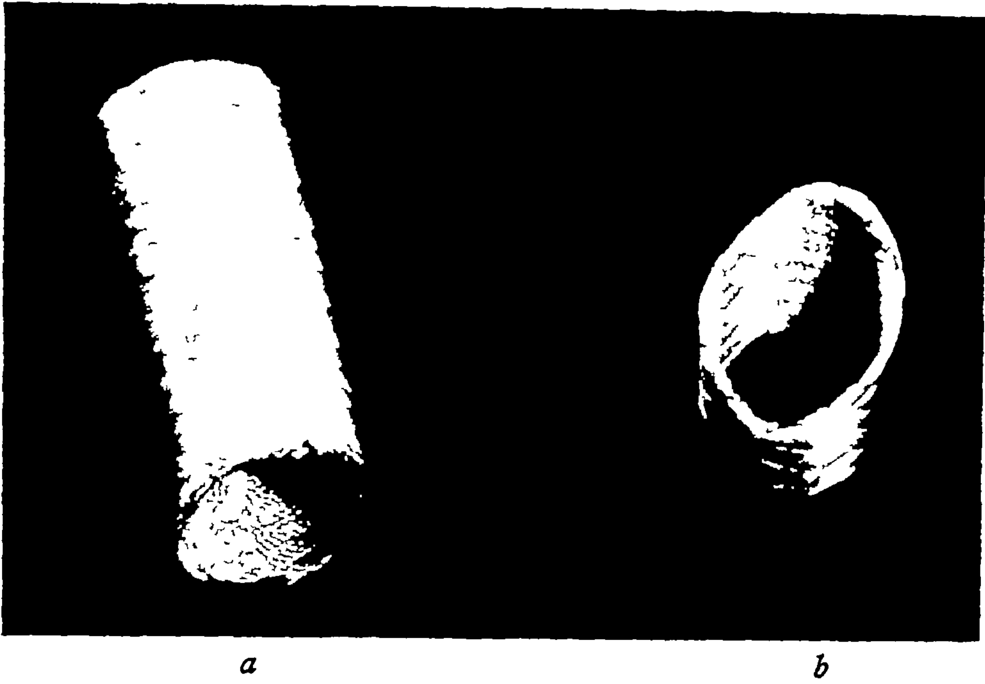
The way in which the covering of the prostheses proceeds is most interesting. It has been our general impression that if a material is used in which there is not laid down any large amount of fibrin, this type of material in general seems to do well. There is a great variety of such materials

In contrast to this, tubes made of solid Lucite, for example, never become covered on the inside by a perceptible layer of tissue. The endothelium will grow up to the edge of the tube and may grow for even 0.5 or 1 mm. onto the lip of the tube, but it will not proceed through the tube, no matter how long one leaves it in place. We have observed these seven or eight years after implantation in the dog.

The variability of the process of covering is always of interest to me, too This varies not only with the weave but seems to vary individually in animals within the same piece of cloth or perhaps cut from a different piece of cloth.

The types of materials which have been used are now manifold, including Dacron, Fiberglass, nylon, Orlon, and many varieties of these cloths, and other polymers of similar nature.

Mr Rob, what do you think about the relationship of arterial homografts—the relation of their length and diameter—to the over all result?



a *b*

Fig 2 A knitted Orlon prosthesis (*a*) before and (*b*) after implantation into the abdominal aorta

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Mr Rob, what do you think about the relationship of arterial homografts—the relation of their length and diameter—to the over all result?

MR. ROB

I think diameter is more important than length. We have found that the results with smaller vessels are less satisfactory. On the other hand, length has an influence. There is very little difference between 6 cm. and 15 cm., but the really long grafts are less satisfactory.

DR. CREECH

I agree in general. I think one of the most important things in the success of an arterial substitute is that there be an adequate lumen distal to the site of implantation. In our experience, every instance of graft thrombosis has resulted from an inadequate lumen distal to the graft preventing the restoration of normal blood flow.

DR. HUFNAGEL

I would like to add one general statement. I think there is a tendency among many individuals to be a little rough with the small vessels. This is a big factor in some of the early failures which one sees reported.

DR. NANSON

I believe the lumen distal to the anastomosis is the important thing, and particularly the nature of the intima just at that point of distal anastomosis. One finds the thrombus in the graft itself, but it originated at the distal line of the anastomosis or even distal to that. So often with grafting in peripheral vessels such as the femoral in arteriosclerosis, it is not only a question of the size of the lumen but also of the poor material encountered in the host.

DR. HUFNAGEL

When using a long graft peripherally, would you electively employ heparin, Dr. Blakemore?

DR. BLAKEMORE

I am afraid I am in a rather confused state about it. It seems to me that one of the more important things to consider is the run-off flow that one gets in the peripheral vessels. Theoretically it should be corrected by a lumbar sympathectomy, but it does not always work.

I think if one does a perfectly good graft but if the rate of flow is not good, thrombosis may occur. Theoretically, heparin would be a good thing to use under these circumstances.

I am unsettled about the question. I have used heparin in a regional manner on many occasions with considerable satisfaction, feeling that I did get a differential that was a safety factor to the patient. In our tests we saw relatively two to three times the heparin level at the spot you wanted it distally, in comparison to the systemic level. I think we need to have a considerable amount of work done on this particular subject in the immediate future if there is to be an improvement in our results with badly diseased peripheral vessels.

DR. SZILAGYI

I believe the diameter of the graft is a very important factor in the result, because beyond a certain size, technical difficulties will arise; but within a very wide margin this usually does not play a great role in the clinical work. We have not encountered difficulty at the lower end of the femoral artery, even though it may have been only 6 or 7 mm. in diameter

As to length, I believe that in very long and relatively narrow grafts the factor of the elasticity becomes important. In dogs we have noticed that when long, plastic, woven grafts have been used, the animals lose their blood pressure considerably This may be very important in the immediate postoperative period.

DR. HUFNAGEL

I believe that emphasizes the problem of having a good bed peripherally, or some place for the blood to go Certainly that is one factor which is not directly under the surgeon's control.

I wonder if Dr Creech would like to say something about heterografts.

DR. CREECH

Arterial heterografts preserved by formalin fixation or by freeze-drying



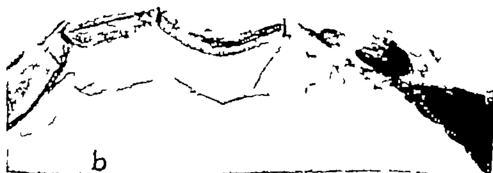


FIG. 1 *a*, Photograph showing large aneurysm which has formed in an experimental heterograft 23 months after transplantation. *b*, Photomicrograph of section taken from the wall of the aneurysm showing thinning and disappearance of the elastic fibers of the media.

have been used experimentally in dogs with generally unsatisfactory results. Degenerative changes in the wall of the heterograft occurred in a majority of instances and consisted of (1) fragmentation and actual loss of elastic fibers with resulting aneurysm formation (Fig. 1), or (2) hyalin change and calcium deposition resulting in rigidity of the graft, and in some instances thrombosis. Only one heterograft has been used clinically, to replace an external iliac artery, and this showed significant dilatation five months after implantation. On the basis of this experience we are of the opinion that arterial heterografts are not satisfactory as vascular substitutes.

DR. HUFNAGEL

We can get a little disagreement at this point. I have not been quite as unhappy as Dr. Creech has been about heterografts In our earlier work, we felt that this was going along quite well I am sure there is no question but that they do show more degenerative changes than homografts, but we have certainly not found them to the same degree as others have We have some animals living for three and a half years after implantation, they appear normal, and on excision at intervals, the total wall has been found to have been well maintained

This question must be reinvestigated in terms of exact techniques. Also, it is worth while to point out that when one speaks of lyophilization, for example, one is not speaking of a process—he is speaking of hundreds of processes The exact conditions under which lyophilization is done may markedly affect the over-all results, and so again I would urge caution in terms of interpreting the problem.

DR. SZILAGYI

Concerning heterografts, we have had some laboratory experience with them, and we had a standardized technique We used the preparation McCuen described, inserting very long segments—15 to 25 cm in length. These were grafts taken from the common carotid of the calf, treated with beta-propiolactone and lyophilized by what I considered to be a very good standard method by one of the excellent local pharmaceutical companies.

The results in these grafts have been completely unpredictable. Some dogs have now lived for almost eight months and are perfectly fine, others have developed fatal hemorrhages.

We find that the heterograft behaves in perhaps one of three ways: First, it may be well accepted, but there is so much reaction around it that eventually it becomes useless. Second, it may become completely sequestrated. There is no hemorrhage around the graft, but it is not invaded and it is not replaced This graft lasts as long as its natural strength will survive, and then it will rupture It will still require some observation time before we have the final answer. Third, occasionally it seems to work.

DR. HUFNAGEL

This is a question which might concern the panel as a whole If you were

going to start a vascular service in a community without major equipment and governmental funds, what method would you use?

MR. ROB

I would use plastic cloth for the large vessels like the aorta, and I would have the simple freeze-drier, which is cheap, for the smaller ones

DR. NANSON

I would go along with Mr Rob's answer, because I am in such a community up in the frozen North. It is hard to get grafts, and that is why I have been so particularly interested in the plastics

I would go along with the plastic materials for the larger vessels, and I would use preserved grafts for the others, and the freeze-drying technique. We are lucky in that we have a bacteriology department which often does freeze-drying

There is one other point I would like to make, and that concerns the value of the plastics because of the extreme ease of sterilization. You can simply boil the nylon, and if you don't have the right size at the moment you can go outside the operating room and make one of the proper size.

DR. SZILAGYI

Plastics sound very attractive even now, but they are not easy to work with. On the other hand, good homografts can be handled with facility I am convinced that any hospital having 200 beds or more, using a simple technique of sterilization, and spreading some local propaganda so that grafts can be obtained, can have this type of work.

How do you preserve the graft? Perhaps the best way is to lyophilize it, but even the cheapest lyophilization machine on the market now costs around $800 Without that, I don t believe one can have a steady supply of any type of grafts at the present time.

DR. HUFNAGEL

If one must avoid the expense of setting up a lyophilization process, one does have the quick-freezing method. If we are working in a single institution the vessels can be kept frozen in that institution very well. It means merely that one must have a CO_2 refrigerator or icebox which can be made for the cost of the insulation and the wood. The vessels can be kept there for the price of the dry ice necessary to maintain the refrigerator

That is probably the simplest way, and it is very effective. The problem, as has been pointed out, is to get the grafts, but with the continued education of members of hospital staffs and the public, no doubt we will find that there are grafts available for the patients who need them.

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INDEX OF SUBJECTS